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SURGICAL ASPECTS OF THE EPILEPSY PROBLEM

COBB PILCHER, M. D.

Nashville

The patient, a victim of the dread falling sickness, lay flat on his face on the beaten earth... The surgeon... held his patient's head, fixed as in a vice, between his powerful, hairy thighs... The falling sickness was always due to a demon entrapped in the skull... The treatment... was a perfectly rational one...

The surgeon was scratching busily with a strong, sharp-pointed flint... a shallow groove in the white bone... soon converted... into a basin-shaped ellipse... The demon had escaped...

Trepanning for the release of demons is the earliest surgical operation of which any evidence remains...

—HARVEY GRAHAM[1]

INTRODUCTION

BEFORE we smile at the superstitious thinking and primitive methods of the Neolithic surgeon described above, let us not forget that in this country alone no fewer than 500,000 epileptics even now have unexorcised demons or that numerous equally illogical surgical procedures are carried out on many of them at the present time. For idiopathic epilepsy is still an incurable disease of unknown cause. Some progress is being made, it is true. Epileptic colonies have been established. Electro-encephalography has shed some light upon the mechanism of the convulsive seizures.[2,3] A new anti-convulsant drug has been discovered.[4,5] But the cause of the condition remains elusive and its therapy is entirely palliative.

Fortunately, however, another aspect of the problem presents a far brighter outlook. The convulsive state is not necessarily synonymous with idiopathic epilepsy. Rather, it represents what Lennox, Cobb and their associates[6,7,8] have called "cerebral dysrhythmia"

From the Department of Surgery, Vanderbilt University School of Medicine.

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and this condition has many possible causes. Many of these causes are amenable to appropriate therapeutic measures. (Summaries of recent publications in this field have been presented in two other papers.[9,10]) It is, therefore, imperative that every patient suffering from convulsions be thoroughly investigated in order to detect some remediable causative condition. The alternative diagnosis of idiopathic epilepsy, if correct, automatically condemns the patient to a lifetime of convulsions and, in many cases, progressive mental deterioration.

It is perhaps appropriate that this subject be presented in Birmingham for here was discovered one of the important causes of convulsive seizures. When Dr. Seale Harris described the clinical syndrome of hyperinsulinism,[11,12,13] he removed, at one stroke, a large number of epileptics from the incurable category. Further investigation has fully confirmed his original opinion.[14]

Similarly, orthostatic (or postural) hypotension, hypersensitive carotid sinus and some forms of hypoparathyroid tetany are sometimes mistaken for epilepsy and should always be looked for in patients suspected of having epilepsy. The occurrence of convulsions in the toxemias of pregnancy, uremia, the fevers of childhood and hypertensive cerebral crises is widely recognized, but even here an incorrect diagnosis may be made.

The particular purpose of this paper, however, is to emphasize and illustrate the existence of a number of organic conditions which cause convulsive seizures and which are susceptible of direct surgical attack upon the cerebral lesion.

Intracranial Tumors

It has long been recognized that many brain tumors are associated with convulsions and numerous comprehensive papers on the subject have been published.[10,15,16,17] In the author's clinic, 30 per cent of the patients with brain tumors have had convulsions. In many instances, these convulsions may be the first, or sometimes the only, symptom. The diagnosis of idiopathic epilepsy is made all too frequently in such cases and operation delayed until the lesion has progressed to inoperable proportions.

In most cases, the convulsions are of Jacksonian, or focal, type. The attacks begin with tonic or clonic movements in one particular muscle group and follow a typical pattern of spread to other parts of the same side of the body and perhaps to the other side as well. On the other hand, the seizures may be generalized and present no focal features whatever. The following three cases illustrate the two types of attack.

CASE 1 (V. U. Hosp. No. 67303). The patient, a 32 year old white housewife, was said to have had syphilis with intensive therapy during her only pregnancy twelve years before admission. During early labor she had a series of convulsions of unknown type. Six weeks after delivery, she began to have typical Jacksonian seizures beginning in the left foot. Each attack was inaugurated by tingling in the left foot and hand, followed at once by clonic jerking of the left foot, spreading rapidly up the leg, into the left upper extremity and left face and often then becoming generalized. Loss of consciousness accompanied most attacks.

These seizures occurred at long intervals for seven years. She was then symptom-free for four years. One year prior to admission, the attacks returned with increasing frequency. The focal pattern was the same. She noticed progressing weakness of the left leg and, later, the left arm. She had occasional headaches.

On examination, there was no evidence of increased intracranial pressure, but she had a spastic left hemiparesis with hyperactive deep reflexes and positive Babinski reflex and slightly diminished pain sensation on the left. The Wassermann reaction was negative in the blood and spinal fluid, and the spinal fluid pressure was normal.

At operation, Nov. 5, 1934, a large partly cystic tumor was removed from the region immediately posterior to the leg area of the right motor cortex. It proved to be the type of very slow-growing glioma called spongioblastoma polare. The hemiplegia was temporarily worse after operation, but rapidly cleared up. She has been symptom-free until the present time.

The amazing feature in this case is the long duration of symptoms. It is also surprising that the tumor showed no calcification. The following similar case was of much shorter duration.

CASE 2 (V. U. H. No. 101964). The patient was a 41 year old white woman whose first symptom was a classical Jacksonian convulsion 10 months before admission. The attack began with clonic jerking of the left hand which spread up the arm, to the trunk, left lower extremity and left face. After the attack the left arm was weak for a few minutes. She had seven similar attacks, with loss of consciousness in only two of them, in the ensuing months. She had been treated for epilepsy and for menstrual dysfunction.

There were no neurologic signs except slight weakness of the left arm and overactive left knee-jerk. The spinal fluid pressure was normal. Roentgenograms showed a small area of "moth-eaten" bone in the right frontoparietal region.

At operation, Sept. 18, 1939, a large meningioma was removed from just in front of the right arm area (fig. 1, a). The overlying dura and bone which had been invaded by the tumor were also removed and a fascial transplant placed in the defect. Postoperative paralysis of the left arm rapidly cleared up. No convulsions have occurred since operation.

Both of the cases just described had focal convulsions, but had been diagnosed epilepsy. Case 3, on the other hand, presented entirely generalized seizures.

CASE 3 (V. U. H. No. 85462). This 36 year old merchant had had convulsions for nearly three years. The attacks began with fleeting dizziness, followed by loss of consciousness and general convulsive movements. He also had transitory "blank spells" suggestive of petit mal. The diagnosis of epilepsy had been made by every physician who saw him and the following therapeutic procedures had been performed: extraction of all upper teeth, two operations on the paranasal sinuses, cholecystectomy, appendectomy and treatment for spastic colon. For several months before admission, he had had intermittent pain in the right side of the face.

There were no neurologic signs except slight left facial weakness and slight hypersensitivity of the right side of the face. The spinal fluid pressure was 340 mm. of water. Ventriculograms showed displacement of the ventricular system to the left.

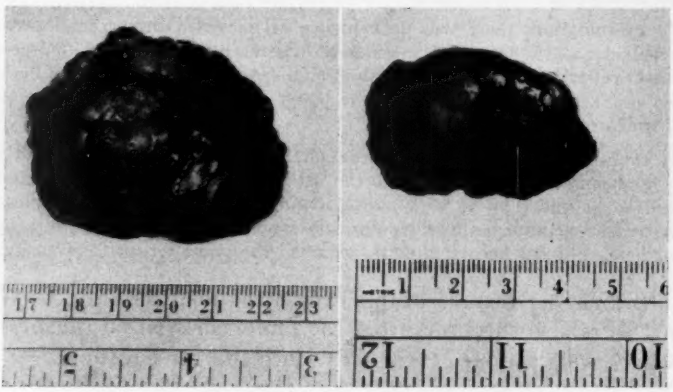

Fig. 1. Meningiomas removed from (a) Case 2, (b) Case 3.

At operation, June 4, 1937, a meningioma arising from the right sphenoid ridge was completely removed (fig. 1, b). The patient returned to work a month after leaving the hospital. He has had two fleeting "dreamy feelings" in the three years since operation, but no other symptoms.

It is of interest that all three of these patients were thought to have idiopathic epilepsy, that none of the three showed choking of the discs and that only one of the three showed any elevation of spinal fluid pressure.

BRAIN ABSCESS

One third of all patients with brain abscesses have convulsions. Furthermore, since abscess of the brain may develop as a blood-borne infection from a distant and sometimes undiscovered focus, the diagnosis may be easily overlooked. Case 4 admirably illustrates this point.

CASE 4 (V. U. H. No. 96017). This patient was admitted at the age of 13 months, six weeks after a fall which seemed to have no immediate harmful

effects. A week after the fall, weakness of the left leg was noticed, and, three days later, she had the first of a series of convulsions always beginning in the left arm and spreading to the left leg and face. There was no history of ear, mastoid or sinus disease or of infection elsewhere. On admission, she had choked discs, and a left hemiparesis. Ventriculograms showed displacement of the ventricles to the left and a filling defect in the body of the right ventricle. It was thought that the child had a tumor or hematoma.

At operation, Jan. 5, 1939, the tragedy of opening widely into an abscess was narrowly averted by the insertion of a needle before the dura was opened. A huge right frontoparietal abscess was entered and partly evacuated by aspira-

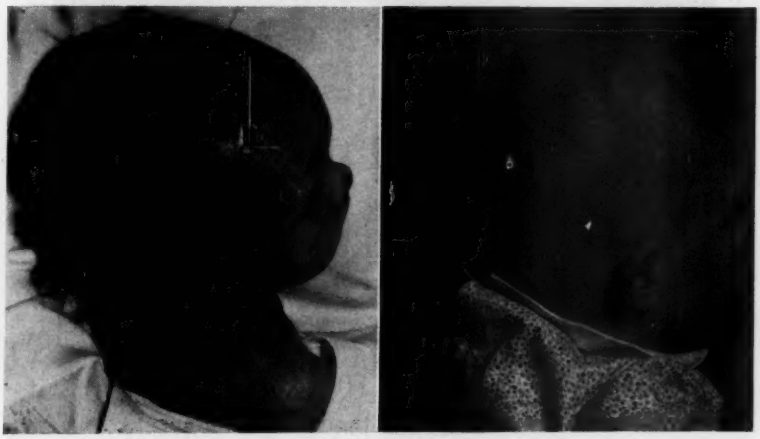

Fig. 2. Case 4. (a) A small cerebral fungus is seen. (b) The fungus has retracted and the wound is almost healed.

tion. Cultures showed a hemolytic streptococcus, but vigorous sulfanilamide therapy was of no benefit (as is frequently true of encapsulated infections). It was necessary to establish open drainage and this proved efficacious, the great cavity healing rapidly and completely (fig. 2). No symptoms have presented since her discharge.

Granulomas

The localized cerebral lesions of syphilis and tuberculosis should be remembered as possible causes of convulsions. Both are very resistant to the usual methods of therapy and frequently require surgical removal. A typical case may be cited.

Case 5 (V. U. H. No. 93920). The patient, a 34 year old housewife, was in the hospital for treatment of a tuberculous sinus of the chest wall by Dr. K. M. Larsen when a history was obtained of attacks of clonic convulsive movements of the right hand. Weakness and spasticity of the hand and arm soon appeared and began to involve the right face and leg.

At operation, Nov. 4, 1938, a large tuberculoma, lying directly in the arm area of the cortex, was removed *en masse*. She recovered promptly and the use of the paralyzed extremity gradually returned (fig. 3).

Fig. 3. Case 5. Photograph of patient 12 months after removal of tuberculoma.

Subcortical Hematoma

It is not commonly recognized that both spontaneous and traumatic hemorrhage may occur in the subcortical region of the brain and that such hematomas are likely to become encapsulated and to produce irritative symptoms long after the original episode. The following two cases illustrate the early and late appearance respectively of convulsions in such cases.

Case 6 (V. U. H. No. 87910). This 38 year old woman had a sudden loss of consciousness with right hemiplegia and aphasia nine months before admission. She was thought to have had an apoplectic "stroke" due to hemorrhage or thrombosis in the internal capsule. Speech and strength in the paralyzed extremities slowly improved, but, after six months, she began to have focal convulsive seizures beginning in the right hand. On admission, she had a partial aphasia and spastic right hemiparesis without pressure signs.

At operation, Oct. 14, 1937, an encapsulated hematoma containing old liquefied blood was found. Both contents and capsule were completely removed and she recovered rapidly both from the operation and from the pre-existing symptoms, none of which has recurred.

The occurrence of focal convulsions in this case clearly indicated that the lesion was in or near the motor cortex; and hence was amenable to surgical attack.

CASE 7 (V. U. H. No. 83636). This 25 year old electrician was said to have sustained a fractured skull at the age of 6. There were no residual symptoms and he enjoyed normal health until the age of 25, four months before admission. At this time, he suddenly had a violent generalized convulsion, with prolonged loss of consciousness. Two similar attacks followed. His neurologic examination and spinal puncture disclosed no abnormalities whatever, but stereoscopic roentgenograms showed an irregular patch of calcification in the right parietal region.

At operation, March 1, 1937, an old encysted hematoma, with thick, partly calcified capsule, was removed. His recovery was complicated by a postoperative, extradural hemorrhage necessitating reopening of the wound but was otherwise complete. In the succeeding three years, he has had two transitory "blank spells," without convulsive movements. Both of these attacks occurred when he had been working overtime and was greatly fatigued.

DEPRESSED FRACTURE

Not only can depressed fragments of the skull cause the subsequent appearance of convulsions, but the presence of the fracture itself, or even the history of injury, may be overlooked as the following cases show.

CASE 8 (V. U. H. No. 83843). A 21 year old girl was admitted because of two generalized convulsions without focal features or residual symptoms of any kind. Only after roentgenologic demonstration of a depressed fracture in the right parietal region did she recall striking her head sharply on the edge of a window a month before the first convulsion.

CASE 9 (V. U. H. No. 85974). This 37 year old salesman was rendered unconscious by a blow on the head 2½ years before admission. A laceration of the scalp in the right parietal region was sutured and healed promptly. A year later he had the first of several convulsive seizures, accurate description of which could not be obtained. Roentgenograms and encephalograms made elsewhere showed no cause for the attacks. There were no neurologic signs, but a tangential roentgenogram, made with the x-rays passing through the plane of the surface of the skull at the site of injury, showed a depressed fracture.

These two cases demonstrate clearly the wisdom of careful search for a focus of irritation in any patient with convulsions. The first of them had to have a cortical scar excised in addition to the depressed bone and has had two convulsions in the two years since this was done. The second has been relieved of attacks for three years by simple removal of the bone.

CEREBRAL CICATRIX

"Traumatic epilepsy" is the term frequently applied to the convulsive seizures associated with cicatricial contracture of the cerebral cortex.[18] The mechanism by which such scars produce convulsions is not clearly understood. It is probable that direct mechanical

irritation by contracture[10] and impairment of local blood supply[19,20,21] play important roles. The incidence of such traumatic epilepsy is exceedingly high—65 per cent in my series of cortical scars.

The results of surgery in these cases is less satisfactory than in any other condition associated with convulsions for which surgery is employed. True most cases are improved by excision of the scar and a moderate number are completely relieved of attacks, but, in many instances, the cicatrix is too extensive for removal or its location is such that complete removal is inadvisable.

Innumerable cases might be cited, but a brief account of one instance in which the cicatrix had probably been present since birth will be adequate for the present purpose.

CASE 10 (V. U. H. No. 54263). The patient, a 14 year old white boy, was born at full term. His mother died in convulsions at the time of his birth. He was "cross-eyed" from birth. Other details could not be elicited. At the age of 9, he began to have convulsions which sometimes began in the right arm and which persisted with gradually increasing frequency until his admission. The only neurologic signs were congenital strabismus, a slight right facial weakness and an inconstant Babinski's sign on the right. However, encephalograms showed a marked dilatation of the posterior portion of the left ventricle which was drawn laterally toward the cortical surface.

At operation, Dec. 14, 1937, there was found a large contracted cicatrix composed of very dense and tough scar tissue. This had distorted the normal structure of the cortex as well as the shape of the ventricle. A radical excision was carried down to the ventricle but some scar was necessarily left behind in the region of the motor cortex. Recently, over two years after operation, he had the first convulsion since leaving the hospital. This is contrasted with the five to ten *daily* attacks occurring before operation.

JACKSONIAN EPILEPSY WITHOUT DEMONSTRABLE LESION

Thanks to the pioneering work of Sachs,[22] Penfield,[23,24] and others, recent years have seen an increasing number of patients with no visible cerebral lesion relieved of convulsive attacks by appropriate cortical excisions. When the seizures have a focal pattern, even though no evidence of tumor, scar or other lesion can be demonstrated before or at operation, the focus of irritation may be located by electrical stimulation of the cortex and excised. The extreme importance of obtaining an accurate account of the seizures or, preferably, of seeing an attack is self-evident. The following cases demonstrate what may be accomplished in such cases.

CASE 11 (V. U. H. No. 95481). The patient was an 18 year old girl who gave a history of seizures for eight years, occurring as often as several times daily. The attacks began with fluttering of the right eyelid followed by clonic movements of the right hand. There was no evidence of increased pressure, encephalograms were normal and at operation (Nov. 28, 1938), the left

cerebral cortex appeared entirely normal. No sign of a subcortical lesion could be discovered. By electrical exploration an area was found in the motor cortex, stimulation of which reproduced the exact pattern of the patient's attacks. This area of the cortex was excised and no attacks have occurred since discharge from the hospital. The temporary aphasia and paralysis of the right face and arm gradually disappeared completely.

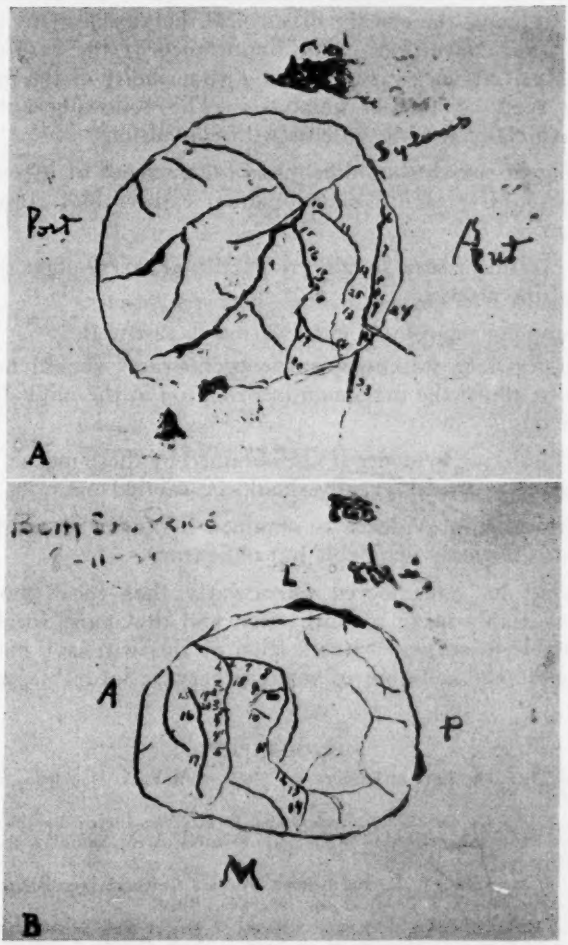

Fig. 4. Photographs of cortical maps made at operation on (a) Case 11, (b) Case 12. These maps are traced on sterilized cellophane placed upon the exposed brain. Each number represents a point electrically stimulated.

CASE 12 (V. U. H. No. 100885). This 6 year old child had had focal attacks for three months. Each attack began with twitching at the left corner of the mouth, then spread to the remaining muscles of the left face and down the left arm and leg, never involving the right side. There were no neurologic

signs and the brain appeared normal at operation. The facial area was located by stimulation and excised (Aug. 11, 1939). The child has had no further attacks.

Discussion

The various conditions and illustrative cases just described are adequate to prove the efficacy of surgical therapy in many patients suffering from convulsions. The importance of the careful diagnostic study of all such cases and the responsibility of the physician therefore need no further emphasis. The following statements become axiomatic for all patients with convulsions.

1. Diligent search should be made for evidence of hyperinsulinism, hypersensitive carotid sinus or other extracranial cause of the convulsions.

2. A careful history should lay particular stress upon the exact pattern of the attacks.

3. Complete neurologic examination is essential.

4. Appropriate stereoscopic roentgenograms should be so selected as to afford the maximum information in the individual case (cf. Case 9).

5. If localizing evidence is not obtained by these means, encephalography or ventriculography should be carried out.

6. If localizing evidence is obtained in any way, exploratory operation is not only justifiable but obligatory.

It should be remembered particularly that focal convulsions almost invariably mean a focal lesion and that most focal lesions are amenable to surgery. Realization of this will save many lives and alleviate the suffering of many otherwise incurable epileptics.

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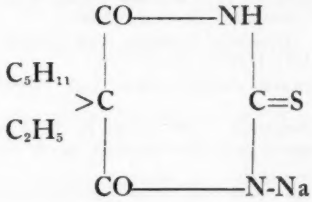
PENTOTHAL SODIUM OXYGEN ANESTHESIA IN MAJOR SURGERY

C. N. CARRAWAY, M. D., F. A. C. S.
Birmingham

OUR first experience with intravenous barbiturate anesthesia was with evipal which was used in minor surgical procedures such as reduction of simple fractures, opening of abscesses and extraction of teeth.

Later pentothal sodium, manufactured by Abbott, became available. It differs from nembutal by the replacement of one atom of oxygen by a sulfur atom on the urea side of the molecule. Its chemical name is sodium-ethyl-(l-methyl butyl)-thio-barbituric acid.



The physiologic action of pentothal sodium in producing anesthesia has not been shown to vary from that of other general anesthetics. The colloid theory of action of anesthetics (Bernard and Bancroft) and the modern lipoid theory of narcosis of Meyer and Overton[1] are the present theories, the latter being generally accepted. The lipoid theory has been expressed in the form of three postulates:

(a) All chemically indifferent substances which are soluble in fats and fat-like bodies must exert a narcotic action on living protoplasm, in so far as they can become distributed in it.

(b) The effect must manifest itself first and most markedly in those cells in which fatty or lipoid substances predominate in the chemical structure and presumably in which they form essential participants of the cell function; namely, in the nerve cells.

(c) The relative efficiency of such narcotic agents must be dependent upon their mechanical affinity for lipoid substances, on the one hand, and for the remaining body constituents, i. e., principally water, on the other hand. Their efficiency is therefore dependent upon the partition coefficient which determines their distribution in a mixture of water and lipoid substances.

According to these conditions, fat solubility is the chief characteristic of the effective narcotic substances of the alcohol group. This is the "fundamental conception" of Meyer and of Overton. This theory has never been disproved.

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No physiologist has ever been able to determine the disposition of pentothal sodium after it is injected into the body. Kohn-Richards states that as yet it has not been recovered from any secretion or excretion of the body if given in therapeutic doses. Barbital and phenobarbital are excreted almost entirely by the kidneys. According to Webster,[1] from 50 to 90 per cent of barbital administered can be recovered in the urine unchanged.

Preoperative Preparation of the Patient

All patients should receive the usual preoperative preparation that is given the evening before operation. It is necessary that the patients receive atropine sulfate about 30 minutes before operation to prevent excessive bronchial and pharyngeal secretion which may bring about laryngeal spasm. This is due to the increased vagus tonus under pentothal anesthesia;[2] thus, the atropine premedication is also experimentally well founded. So far, we have not had an instance of laryngeal spasm.

It is essential that all patients receiving pentothal sodium oxygen anesthesia have an empty stomach or they may vomit at the beginning of the second stage of anesthesia and there is danger of aspirating the vomitus. If the patient's condition demands it, barbiturate compounds can be administered the evening before operation.

The Method of Administration

After the patient has been placed upon the table, the field of operation prepared and the patient draped, the arm to be used for injection is brought out at right angles to the table and attached to the arm rest. The cuff of the sphygmomanometer is applied to the arm and the blood pressure recorded. When the surgeon is ready to make his incision, the anesthetist inserts a small needle (22 gauge) into the vein and begins to administer the solution of pentothal as slowly as possible. The patient is asked to count. Soon the patient ceases counting and often yawns as he falls into a condition resembling natural sleep. There is no excitatory stage. The assistant anesthetist, who is sitting at the patient's head, holds the jaw upward and forward. The oxygen mask is adjusted to the face and continuous administration of oxygen begun. The surgeon tests the sensibility of the patient by pricking the skin. If there is any indication of sensation, the anesthetist slowly injects a few more minims of solution, until the patient is under surgical anesthesia. It takes from one half to three minutes to produce this condition. Then the administration of solution is stopped, but the needle is left in the vein. At this stage the skilled anesthetist begins a careful neurologic examination of the patient, which is continued throughout the operation. If there is the slightest indication that more anesthetic is

needed, as much of the solution is injected slowly as the patient demands to keep him surgically anesthetized.

The strength of the solution used varies from 1.25 to 5 per cent depending on the age, size and physical condition of the patient. The weaker solutions are administered to the very young, to the aged and feeble, and to patients who are in shock.

Picrotoxin is an antidote for barbiturate. In most instances, 2 c.c. of 0.3 per cent solution is given at the end of operation before the needle is removed from the vein. After using picrotoxin in many patients, we are of the opinion that it hastens the recovery from anesthesia.

Patients respond differently to a given dose of pentothal sodium. Only 3 c.c. of 5 per cent solution were required by a patient with a ruptured ectopic pregnancy, who was in shock and required transfusions during the operation, which lasted 20 minutes. We feel that if the patient had been given 6 to 10 c.c. or any amount estimated by body weight, she might have died due to the anesthetic. We have given children weighing 60 to 70 pounds as much as 10 to 12 c.c. for appendectomies. We have seen large individuals who have required only a small amount of drug for a 30 to 40 minute operation, and smaller patients who have taken twice the amount for the same type of operation requiring the same length of time.

We have used most drugs and chemicals that produce general anesthesia, such as ether, nitrous oxide-oxygen, ethylene-oxygen, cyclopropane-oxygen, and also have had experience with spinal and rectal anesthetics, and have found that pentothal sodium oxygen anesthesia gives the most relaxation and causes less interruption during the operation from straining, when the patient reverts to a light stage of anesthesia. Pentothal sodium does not produce sweating and there is no loss of body fluid due to anesthesia.

Systemic Effects

Before we adopted the technic of continuous administration of oxygen during the anesthesia, the respiration became somewhat shallow and quiet, and the patient developed a pallor. In the early part of 1938, I conceived the idea that the continuous administration of oxygen might eliminate this pallor. A nasal catheter was attached to an oxygen tank and inserted into the nose and a continuous flow of oxygen administered to keep the patient well oxygenated. The rate and volume of respiration and color remained about normal. In April 1939, we began to use the B. L. B. mask and bag.

The pulse, determined on every patient before, at intervals dur-

ing and after anesthesia, varies only slightly in rate. The volume is not changed.

The blood pressure, recorded as a routine before, during and after anesthesia varied not more than ten points in any patient. Some other workers have found that patients with hypertension often show a greater fall in blood pressure.

Electrocardiograms were recorded on 65 patients before operation, at intervals during operation, immediately after operation, and from one to ten days following operation. These tracings were examined by Dr. E. Dice Lineberry, Norwood Clinic, Dr. Edgar Hull, Louisiana State University, and Dr. R. Kohn-Richards, Abbott Laboratories. In some instances, slight but insignificant changes were noted.

Routine laboratory work is done on all patients, which includes urinalyses, hemoglobin, erythrocyte, leukocyte, and differential counts, and coagulation and bleeding time. Coagulation and bleeding time are slightly decreased on the average during anesthesia.

Twenty-five patients whose blood chemistry was normal before operation were selected, and the blood sugar, non-protein nitrogen, uric acid and creatinin were determined before and one day after operation. There was no change noted. A large number of patients who had abnormal blood chemistry before operation were selected and the blood chemistry determined before and one day after operation. The results were the same as before operation. However, in ten patients whose blood sugar was normal before operation, there was an average rise of 5 per cent in blood sugar when determined immediately after the anesthetic was discontinued.

The Quick hippuric acid test[3] of liver function was made on many patients. A test was made on the day preceding operation and then daily for ten days following operation. After excluding those patients who had postoperative vomiting, there were left 100 patients on whom all tests were completed. No change in liver function was noted.

The results of the above tests have been reported in detail.[4]

Experimental Results

Using the method of Gregory and Pascoe[5] for determination of liver function by analysis of bile acids from bile drained from the gallbladder, we have studied the effect of repeated doses of pentothal sodium. A dog was anesthetized with pentothal sodium and a mushroom catheter inserted into the gallbladder. After allowing a week for recuperation, an average percentage of bile acids secreted was obtained by analysis of bile on three successive days. Later the dog was anesthetized with pentothal sodium and further analyses

carried out. There was no change noted. These experiments are to be continued.

Under the direction of Dr. R. Kohn-Richards, pharmacologist in the research department of Abbott Laboratories, experimental work has been done on mice, rabbits, dogs and monkeys, to determine the effect of pentothal sodium on the liver. These results will soon be published. With Dr. Kohn-Richards' permission, I quote the following:

We have done a considerable series of experiments with pentothal sodium and find that in mice, necrosis, as reported by Reynolds, has never been observed; however, fatty infiltration of the liver appears within 24 hours but clears within 72 to 96 hours. This is with intraperitoneal injection of pentothal.

Rabbits and dogs do not show any fatty infiltration after one, or even two, doses of pentothal intravenously.

In monkeys, after 24 hours, slight fatty infiltration of the liver, which disappeared rather rapidly, was noted.

The histologic slides of biopsies of the liver made before, immediately after and for several successive days thereafter, showed no necrosis, but did show slight fatty infiltration of the liver. There was no evidence of destruction of cell structure—the cells were large with nucleus pushed to one side.

We feel that our work with monkeys would indicate that there is a mobilization of glycogen from the liver, producing a slight rise in blood sugar during pentothal anesthesia. The glycogen is replaced by fat. This, however, disappears entirely by the end of the third or fourth day after anesthesia.

Dr. Kohn-Richards subjected one dog to approximately sixty pentothal injections under careful control of blood picture, urine, liver function and non-protein nitrogen. At the end of the experiment the blood findings were approximately the same as in the beginning. The urine showed a transitory albuminuria. The blood sugar and non-protein nitrogen were normal at the end of the experiment, as was the bromsulphthalein liver test. Histologic biopsies of the liver which were taken daily during the test showed some fatty infiltration in some of the last biopsies. There are more experiments going on which are not far enough advanced to draw definite conclusions.

It was at one time thought that during general anesthesia, lipoid or fat-like substances were transferred from the brain cells and deposited in the liver and in recovery from anesthesia a reversible action took place. This theory has been disproved, but in all general anesthesia, a varying degree of fatty infiltration of the liver occurs.

Pentothal Sodium Anesthesia in Major Surgery

I will not attempt to discuss the results in every type of major operation performed under pentothal sodium oxygen anesthesia, but will elaborate on a few of the more important ones.

SOME MAJOR OPERATIONS PERFORMED UNDER PENTOTHAL SODIUM ANESTHESIA

Aug. 1936 - Feb. 1940

Thyroidectomy	66
Cholecystectomy	100
Cholecystostomy	9
Appendectomy	1177
Radical mammectomy	20
Gastric resection	23
Resection of peptic ulcer	35
Suturing perforated gastric ulcer	3
Hysterectomy	407

Our method of handling patients with extreme hyperthyroidism is as follows: They are admitted with the understanding that they are going to be operated upon, but they do not know the exact date. They are given hypodermic medication of vitamin B_1 and glucose intravenously, and sometimes iodine preparation (homiodin). After observing the patients for a few days, if we feel that they are in a proper condition for operation, the time of operation is set. The anesthetist goes to the patient's room, leaving the wheel cart outside so that the patient will not become suspicious. Sufficient amount of pentothal solution to produce a light anesthesia is injected. The needle is removed from the vein, and the patient is transferred to the operating room, where everything is in readiness. As soon as the patient is placed on the operating table, continuous administration of oxygen is begun and the field of operation is prepared. The needle is reinserted into the vein and the sensibility of the patient is tested. If necessary, more of the solution is injected gradually and at intervals throughout the operation as is needed. When the patient is sent back to his room, he is put under an oxygen tent. When he begins to react, he is still sufficiently under the effect of pentothal sodium not to be concerned about his condition. We have had less reaction from this anesthetic than from any other. There has been no evidence of postoperative crisis; however, our number of cases is limited. Two patients had enlarged hearts with auricular fibrillation, but they got along surprisingly well.

In most instances, the patients with acute cholecystitis were not seen until after they had been treated at home from two to four days and had gotten progressively worse under palliative treatment and were then referred to the hospital. Because of the perfect relaxation of the patient under pentothal sodium anesthesia, operative conditions were most satisfactory. As far as we were able to determine,

there was no indication of hepatic damage because of the administration of the drug. The jaundice present in some cleared immediately after operation. Most patients were conscious within two or three hours after operation.

The mortality in patients with appendicitis has been reduced since using pentothal sodium anesthesia. In my opinion the advantage has been in the complete relaxation—there is no straining or periods of resistance or the slightest excursion of the intestines into the field of operation. Since using pentothal, we have operated on 1,177 patients primarily for appendicitis. (Appendectomies performed secondarily in combination with some other operation are omitted from this series.) Of these, 205, or 17.4 per cent, were gangrenous; and 90, or 7.6 per cent, were ruptured or abscessed.

We have had six deaths from appendicitis since April, 1936. Three of these were diagnosed definitely as appendicitis and were then operated upon.

CASE 1. A Negro man, aged 38, had a ruptured retrocecal appendix. There was no attempt by nature to wall off the infection. Death, which was caused from spreading peritonitis, occurred on the fifth postoperative day.

CASE 2. A white boy, aged 9, was brought to the hospital in a moribund condition. After consultation it was decided that the child could not live, so we decided to give him the benefit of an operation. He died about 22 hours after admission.

CASE 3. A white girl, aged 16, was admitted to the hospital complaining of a soreness in her side which had begun 7 days previously. There was a definite walled-off abscess at the brim of the pelvis that extended into the pelvis. This was incised and drained. The following morning the patient had a sudden rise of temperature (105.5°). Rales were noted throughout the base of the lungs. She was put under an oxygen tent and given sulfapyridine. Death, which was caused from pneumonia, occurred about 48 hours later.

The other three cases may be summarized as follows:

CASE 4. A white male, aged 50, was admitted in a semiconscious condition. He died six days after admission, no definite diagnosis having been made. Autopsy revealed a ruptured postperitoneal appendix with spreading infection in the postperitoneal tissues.

CASE 5. A Negro woman, aged 56, on examination was found to have a large fluctuating mass in the lower abdomen. The cul-de-sac was punctured and a large amount of pus drained. Four days later she died. Autopsy revealed a gangrenous appendicitis with perforation, cul-de-sac and multiple peritoneal abscesses.

CASE 6. A white man, aged 36, gave a history of having had abdominal pain for five days. He was vomiting fecal material and had all the symptoms of acute intestinal obstruction. A walled-off abscess filling the lower right quadrant was found. A small stab wound was made just inside the crest of the ilium and a large amount of offensive pus, which was under great pressure,

was let out and a drain inserted. The patient continued to vomit fecal material and died two days later. Our diagnosis was death from acute intestinal obstruction probably due to appendiceal abscess. No autopsy was performed.

Fatalities in Patients Not Yet Recovered from the Anesthetic

Up to the present time, we have had three deaths during anesthesia.

Case 1. A white man, aged 64, had an inguinal hernia of several years duration. The hernia became strangulated on June 9, 1939. Although frequent attempts were made by himself and his physician, the hernia could not be reduced. About 48 hours later he was admitted to the hospital for operation. On his arrival his abdomen was distended and rigid, and he was vomiting large quantities of material with fecal odor. Just as he reached the second stage of anesthesia, he vomited a large quantity of this material which was aspirated into his lungs. He became cyanotic, continued to vomit and drowned from his own vomitus in spite of our efforts.

Case 2. A Negro man, aged 51, was brought to the hospital as a result of a crushing laceration to his right hand and wrist. He was given atropine and morphine as a preliminary. He was then given 15 c.c. of 3.75 per cent solution of pentothal, which was administered slowly over a period of ten minutes beginning at 6:55 p.m. The patient's respiration was 20 and shallow; pulse 90, poor volume at the beginning of administration. Heart action and respiration ceased at 7:10 p.m. Epinephrine, coramine, continuous oxygen under pressure, and artificial respiration were administered without effect. We were fortunate to obtain an autopsy on this patient. The findings as reported by Dr. G. S. Graham and Dr. L. C. Posey are as follows:

Anatomic Diagnosis:

1. Crushing laceration of the right hand.
2. Air emboli in coronary veins, pulmonary veins and arteries, and portal veins in the liver.
3. Marked cardiac hypertrophy.
4. Syphilitic aortitis.
5. Generalized arteriosclerosis.

Microscopic Diagnosis:

1. Myocardial fibrosis.
2. Chronic vascular nephritis.
3. Slight passive congestion with fibrosis.
4. Occasional fibrosis of pancreatic islets.

Death, in the opinion of the pathologists, was due to air emboli.

Case 3. A Negro man, aged 35, weighing about 200 pounds, was first seen by the medical department on Aug. 16, 1939. He gave a history of dizziness and pain in epigastrium. He had generalized edema and his blood pressure was 200/130. He was treated by the medical department for essential hypertension. Under rest, his systolic blood pressure came down to 180 and diastolic to 120. X-ray of the chest revealed an enormously enlarged heart, almost filling the lower half of the thoracic cavity.

On Feb. 22, 1940 he was admitted to the hospital complaining of rectal pain. He was found to have an anal fissure. He insisted on having it removed. One of the members of the surgical staff agreed to remove it, after warning the patient of the danger of any type of anesthetic. After receiving 8 c.c. of

5 per cent solution administered as slowly as possible, just as he entered the second stage of anesthesia, his heart action and respiration stopped. Artificial respiration, epinephrine, coramine, picrotoxin and forced inhalation of oxygen were without effect. Autopsy was refused.

His Wassermann was negative, but he gave a history of rheumatic fever and acute nephritis.

It is my opinion that the result would have been the same under any type of anesthesia. The mistake was in using a general anesthetic. It is unfortunate that permission for autopsy could not be obtained.

Summary and Conclusions

Since August, 1936, through February, 1940, we have administered pentothal sodium 5,963 times to patients ranging in age from 2 to 90. This series includes all general surgery, most of the obstetric and neurologic surgery, and that performed in the eye, ear, nose and throat department, except tonsillectomies. There have been a few tonsillectomies performed under pentothal sodium anesthesia, but it has been found that the region of the pharynx which is supplied by the glossopharyngeal, a branch of the fifth, ninth and superior laryngeal nerves, is not affected until a very deep stage of anesthesia.

We have used this type of anesthesia in all types of surgery with all kinds of complications with the most gratifying results. Some of the major surgical procedures are discussed in detail.

Most conditions listed as contraindications to the use of this type of anesthesia have been found to be indications. If there are any contraindications, it is my opinion that they may be certain types of heart conditions aforementioned. However, we have used the anesthetic successfully on many occasions in patients with hypertension and cardiac disease with congestive failure. We have also used it successively in four patients who had intense jaundice.

Pentothal sodium oxygen anesthesia is not foolproof and anyone administering it should be skilled in the method of administration of all types of anesthetics. The anesthetist should be able to gauge and regulate the depth of anesthesia by the neurological signs of the patient.

Norwood Hospital, Birmingham.

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A SIMPLE TECHNIC FOR GLASS BALL IMPLANTATION FOLLOWING ENUCLEATION OF THE EYEBALL

RALPH O. RYCHENER, M. D., F. A. C. S.
Memphis

ENUCLEATION of the eyeball is often mentioned as the most satisfactory of ophthalmologic operations. As a rule, the patient parts with an eye only after a long period of inflammation, pain and disability or because of a disfiguring appendage which is an economic liability and he is therefore happy in his relief from pain or an improvement in his personal appearance. It has been the experience of many ophthalmologists that some of their most grateful patients have been those for whom enucleation was performed. Especially has this been true in the past two decades when the implantation of some foreign substance in Tenon's capsule with an improved cosmetic result has become more common.

Simple enucleation of the globe was devised by Ferrall and Bonnet and is still practiced by many who do not realize the cosmetic advantage of an implantation operation or who are deterred from using the latter because of the numerous complicated technics which have been described. Glass, gold, and silver balls have been implanted in the sclera after evisceration and often result in very happy results. But the intense reaction which follows, often accompanied by loss of the implant and the occasional unhappy complication of sympathetic ophthalmia have brought the Mules' operation into some disrepute. Enucleation has many advantages over evisceration, one of which is the preservation of the globe for pathologic study, which has added so much to our knowledge of disease of the eye.

Frost and Lang in 1886 conceived the idea of implanting some solid substance in Tenon's capsule and various types of foreign bodies and various types of closure of the wound have been advocated by subsequent authors. Some of these substances have been spherical implants of gold, silver, glass, fat, cartilage, fascia, aluminum, paraffin, burned bone, steel mesh, corp, and porcelain. The technics have included separate suturing of the capsule which is later covered with the extraocular muscles, or by complicated closures including capsule, muscles and conjunctiva.

For many years Dr. E. C. Ellett and I have used a method of glass ball implantation which has been satisfactory in our hands and which has the advantage of simplicity as compared to most of the

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technics previously published. There is nothing particularly new in this procedure as it is a combination of the Ferrall-Bonnet and Frost-Lang methods combined with some good advice by Greenwood and Ralston. We have added a few points in technic which have reduced our losses of the implant to a minimum as it has now been more than ten years since we have suffered such a loss. We feel that the operation is so simplified that there is no longer any excuse for doing a simple enucleation except under the most extenuating circumstances.

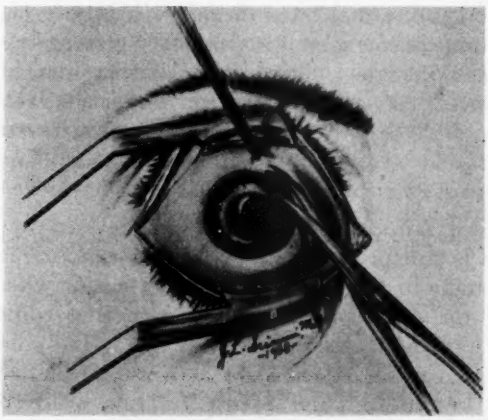

Fig. 1. Incising the conjunctiva.

Practically all of our implantations are done under local anesthesia, the only contraindication to this method being the prohibitive youthfulness of the patient. We give a hypodermic injection of morphine sulphate, grain ¼, and hyoscine hydrobromide, grain 1/200, one hour before the operation. In females of small stature the dose of morphine is cut in half. Occasionally in apprehensive patients some additional sedative from the barbiturate group is given several hours before operation. The usual routine administration of six drops of 4 per cent cocaine solution is carried out, and then there is given a ciliary ganglion injection of 1 per cent procaine hydrochloride solution with epinephrine, usually from 2 to 4 cc., and always enough to produce a slight proptosis of the globe. Under this procedure practically no patient has experienced any pain or even realized that an operation has been performed. No undue hemorrhage has been encountered following this technic.

The conjunctiva is cut by closely circumscribing the limbus, undermined, and the respective recti tendons severed (fig. 1).

The undermining which separates all the tissues from the globe

back to the equator, is done by putting the closed scissors in and opening them, a satisfactory method which we have not seen generally used by others (fig 2).

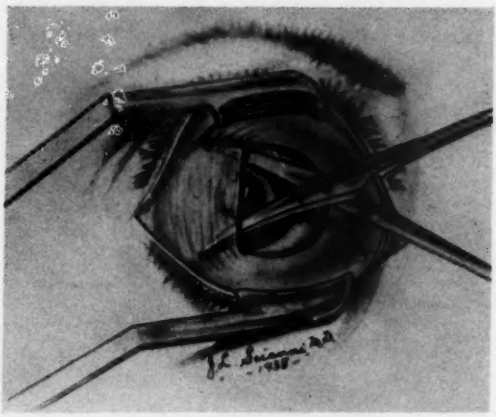

Fig. 2. Separating the conjunctiva from the globe.

The internus is cut on the mesial side of the elevating hook, the remaining three severed close to the ball. The internus stump is grasped with a small curved hemostat to give a firm hold and control of the globe (fig. 3).

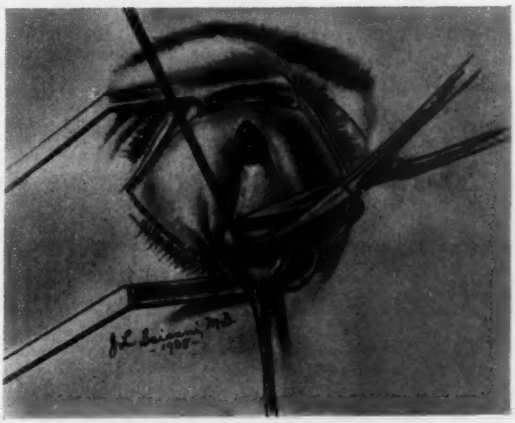

Fig. 3. Cutting the muscle from the globe.

The oblique tendons can usually be isolated and severed at this time which enhances proptosis and delivery of the globe although these muscles can be left attached until after severance of the optic nerve. The globe is then proptosed by depressing the speculum,

rotated externally and the nerve cut from the nasal side. The oblique tendons and accompanying tissues if not already severed are dissected away close to the sclera and nerve, and a hot saline pack inserted with bayonet forceps to the apex of the orbit, and held in position by firm pressure till all bleeding has ceased.

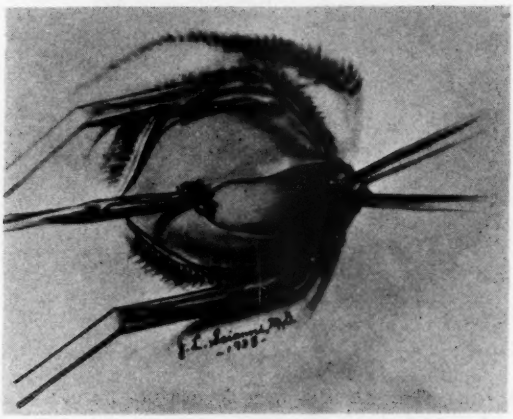

Fig. 4. Grasping the stump of the internal rectus tendon with a hemostat. Cutting the optic nerve after locating it by feeling with the point of the scissors.

Dr. D. H. Anthony of Memphis has devised an olive-tipped compressor which fits snugly into the apex of the orbit and successfully controls the hemorrhage which usually accompanies section of the nerve. With the introduction of this instrument and firm compression, it may not be necessary to introduce a hot pack.

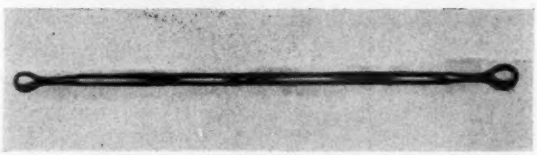

Fig. 5. The enucleation orbital compressor (Anthony).

The capsule is then picked up by four mosquito forceps, as illustrated, and it is always surprising to see what a complete, strong, tenacious membrane the shining capsule is (fig. 6).

With some convenient instrument such as a Mules repositor, a suitable-sized glass ball, usually 16 or 18 mm. is placed in the capsule, firmly depressed and held in position by a strabismus hook, which serves not only to keep the ball out of the way while

closing the capsule, but helps also to prevent bleeding or oozing behind the ball (fig. 7).

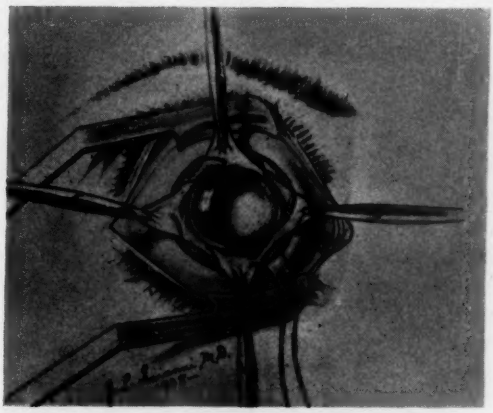

Fig. 6. Exposing the cavity exposed by Tenon's capsule. The glass ball being introduced.

The capsule is closed with a purse string suture of 00 chromic catgut. The conjunctiva is closed with a horizontally running suture of silk, the edges being left long to facilitate removal and a firm pressure head bandage is applied (fig. 8).

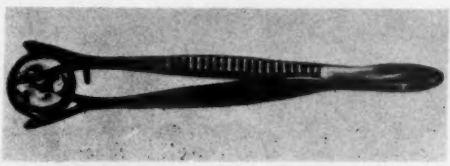

Fig. 7. The glass ball held in a double fixation forcep, which is a suitable instrument for holding it.

From some of our subsequent results we believe this pressure to be a very important factor in securing retention of the ball. The patient is kept in bed for twenty-four hours, allowed up and about thereafter, and after seventy-two hours the pressure bandage is taken off. The conjunctival suture is removed by cutting it centrally and withdrawing the two ends left long at operation. The patient leaves the hospital at this time with only an ordinary eye dressing, is seen occasionally for cleansing purposes, and as a rule is ready for a prosthesis at the end of two weeks (fig. 9).

In omitting special suture of the muscles, we are influenced by the fact that the muscles do not normally join together over the eye ball, but are attached to the globe a little in advance of the

equator. The purse string suture in the capsule brings the muscles sufficiently far forward for them to impart the desired motion to the glass ball, and to unite them over the ball would seem likely

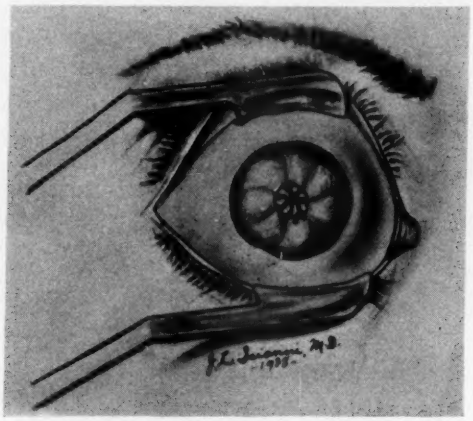

Fig. 8. The capsule, with the muscles, is closed with catgut suture.

to pull the ball so far back in the orbit that much of its value as a support to the prosthesis would be lost. This may account for the experience of some surgeons who have been troubled by displacement of their implants into other parts of the orbit.

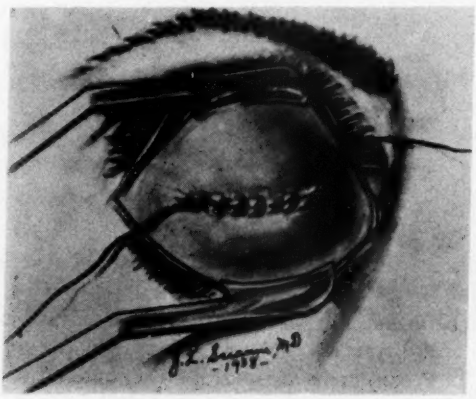

Fig. 9. Closure of the conjunctiva.

The minor developments that we have added to the technic of implantation following enucleation which we feel are responsible for our continued success with this operation are:

a. the use of glass balls with a maximum diameter of 18 mm.,
b. a purse string catgut suture in Tenon's capsule, and
c. a pressure bandage for 72 hours following operation.

Dr. Greenwood advocated the introduction of glassballs of 20 to 24 mm. in diameter and for many years we followed that advice. Our losses of the implant all occurred during the period in which we used the large balls and we feel were to be directly attributed to their size, as we have had no losses since reducing our implants to a maximum diameter of 18 mm. Indeed there seems to us to be a mathematical basis for choosing the smaller ball. Tenon's capsule closely invaginates the eyeball from the circumference of the cornea to the entrance of the optic nerve. The average sized adult eye with a diameter of 24 mm. has a circumference of 75.8 mm. Subtracting 12 mm. for the loss of the cornea leaves an intracapsular circumference of 63.8 mm. A glass ball of 20 mm. diameter has a circumference of 62.8 mm. while one of 18 mm. has a circumference of 57 mm. The latter allows enough leeway for satisfactory introduction of the purse string suture and affords capsular union without excessive intracapsular pressure. A sixteen to eighteen mm. ball, with a carefully selected "shell eye," and not a "reform eye," gives us the best cosmetic results. In conversation with a prominent maker of artificial eyes who also supplied the balls for implantation, we were surprised to learn that he advocated smaller balls, even to 8 mm. in diameter. He uses "reform" eyes, which, of course, require more room than the shell eyes do.

The use of the catgut suture in Tenon's capsule avoids the necessity for later removal, a condition which I experienced frequently in my early days when my chiefs-of-staff used interrupted silk sutures in closing the capsule and it was my lot to remove subconjunctival silk sutures for months thereafter.

The application of constant and continued pressure over the implant causes direct and rapid union of the wound and prevents the accumulation of blood and serum behind the implant, thereby preventing its expulsion by intracapsular pressure.

Some of my colleagues have discredited the implantation operation because of late displacement or wandering of the implant into other parts of the orbit. We have never as yet had a ball become displaced or slip out between the muscles into the orbit, and it is difficult to see why this should happen unless the capsule is cut or torn in some manner during the operation. There is little reason for such an occurrence to happen to anyone familiar with the anatomy of the region. It is conceivable that the implant might pass backward in the muscular core so far as to be of little use in supporting an ordinary prosthesis. Even so, it would be possible to construct a heavier prosthesis in the form of a "reform eye" which would still give an improved cosmetic appearance.

THE SPLENIC ANEMIAS

J. D. Rives, M. D.

From the Department of Surgery, Louisiana State University School of Medicine, and the Charity Hospital of Louisiana

New Orleans

THE term splenic anemia was first used by Gretzel in 1866 in the report of a case of splenomegaly with anemia not associated with leukemia. In 1882 Banti,[1] in a monograph on splenic anemia, first reported the splenomegaly with cirrhosis of the liver that has become identified with his name and which he described more completely in 1894 and in 1898. The disease as described by him was characterized clinically, in its first stage, by splenomegaly, hypochromic anemia, and a normal leukocyte count, and, in its later stages, by the additional development of atrophic cirrhosis of the liver with ascites and, occasionally, jaundice. Pathologically, it was characterized by fibrosis of the malpighian corpuscles and the reticulum (fibro-adenia), absence of abnormal erythrocyte destruction in the spleen, degenerative changes with plaque formation in the splenic vein and in that part of the portal vein traversed by the blood from the spleen; and, in the late stages, by atrophic cirrhosis of the liver with its resultant portal congestion and ascites. He believed that the splenomegaly was caused by some unknown toxin and that the spleen itself, due to its diseased state, elaborated a toxic substance that caused degenerative changes in its veins and also atrophic cirrhosis of the liver. He proposed this theory because he had been unable to find similar venous changes in the rest of the portal system, because he believed that the splenomegaly always preceded the cirrhosis, and because splenectomy had in several instances arrested the progress of the disease.

Contrary to popular belief, Banti did not consider his disease an entity. In 1894 he stated, "Splenomegaly with hepatic cirrhosis ought not to be looked upon as forming a separate morbid entity, but rather as a more advanced stage of splenic anemia." It was logical, therefore, that Osler,[2] when he introduced the name splenic anemia into the English language with papers in 1900 and in 1902, included Banti's disease within this more comprehensive category. Osler defined splenic anemia as,

> a chronic affection, probably an intoxication of unknown origin, characterized by progressive enlargement of the spleen which cannot be correlated with any known cause, as malaria, leukemia, syphilis, cirrhosis of the liver, etc. (primary splenomegaly), a marked tendency to hemorrhage, particularly from the stomach, and, in many cases, a terminal stage with cirrhosis of the liver, jaundice, and ascites (Banti's disease).

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He recognized the facts that syphilis of the liver may cause a splenomegaly that is clinically indistinguishable from that of splenic anemia and that the same syndrome may result from a variety of causes.

It is apparent from a study of Osler's reports that cases of both Gaucher's disease and hemolytic jaundice were included in his collection. It was not until 1907, when Chauffard discovered the increased fragility of the red blood cells in hemolytic jaundice, that this disease, which had been described by Claude Wilson in 1890, was set apart from the splenic anemias; Gaucher's disease, first described in 1882, was finally identified as a separate entity by Mandelbaum in 1912. (See Moynihan.[3])

In the early years of this century, Banti's idea that splenomegaly was the primary change in splenic anemia seems to have been rather generally accepted, but subsequent observations have led many to doubt this. It has been found that the pathologic changes as well as the clinical picture of this syndrome may result from, or be associated with, disease of the liver due to a wide variety of causes including syphilis, hydatid disease, and schistosomiasis. This has led to the belief that the liver damage is the primary cause, and Campbell[4] and McIntosh (quoted by Campbell), in particular, have been so impressed with the similarity between the splenomegaly and associated changes occurring in schistosomiasis and those occurring in Banti's syndrome that they suggest that biopsies of the liver might show schistosomiasis in all cases. However, Whipple[5] and his co-workers have failed to confirm this suspicion, although they have been able to cause progressive splenomegaly by artificially produced cirrhosis of the liver. McMichael,[6] after a most admirable pathologic and clinical study of splenic anemia, is still convinced that hepatitis is the primary etiologic factor in spite of his own observation that great enlargement of the spleen may occur before gross changes in the liver are evident.

Many observers have been impressed by the fact that portal congestion is a constant feature in the splenic anemias, and numerous attempts have been made to prove that the splenomegaly is a result of portal hypertension. This belief is supported by the facts that: first, portal hypertension has been proved to be present in all cases tested (Rousselot[7]); second, mechanical obstruction of the portal or splenic veins has been found in 70 per cent of all cases examined by Whipple and Rousselot[5]; third, all cases of thrombosis of the splenic or portal veins and cavernous transformation of the portal vein cause, or are associated with, the syndrome of Banti; and, fourth, that in cirrhosis of the liver, from whatever cause, the spleen is usually enlarged. But, the fact that portal hypertension

is associated with splenic anemia does not prove that it is the cause of it. As a matter of fact, active congestion of the spleen might conceivably cause the hypertension. In the second place, if mechanical obstruction (including cirrhosis of the liver) is the cause of the portal obstruction and secondarily of the splenomegaly it should be found in all cases, and it should be possible to reproduce the disease by experimental means. But, it has long been known (Pibram, 1902; Warthin, 1910, both quoted by McMichael[6]) that complete occlusion of the splenic vein produces a temporary enlargement of the spleen not greater than three times normal size (about 450 Gm.). (It should be noted that this enlargement, due to passive congestion alone, is much less than the average enlargement of the spleen in splenic anemia—approximately 800 Gm.)

Ultimately, complete occlusion of the splenic vein results in atrophy and both Jaeger (quoted by McMichael[6]) and Menon[8] have failed to produce gross or progressive splenomegaly by long-continued partial occlusion. Menon concludes that

> ...venous congestion of whatever type, is followed by immediate and progressive enlargement which lasts only for a short time and is gradually followed by shrinkage and fibrosis. Hyperplastic reactions do not play any part in the experimental animals after portal obstruction.

Thromboses of the splenic and portal veins may be and probably are secondary to the degenerative changes in these veins described by Banti and confirmed by many subsequent investigators. There is no sound reason for considering them the primary cause of the associated splenomegaly. It is true that idiopathic cirrhosis of the liver is usually associated with splenomegaly, but the degree of enlargement is not often greater than that which results from ligation of the splenic vein. If cirrhosis of the liver causes Banti's syndrome by means of the resultant portal hypertension, all cases of advanced cirrhosis should present this syndrome.

With this idea in mind, I have studied the autopsy records of 51 consecutive cases of portal cirrhosis of the liver at the Charity Hospital. In 34 out of 51 cases (67 per cent), the spleen weighed over 150 Gm., but in only 5 out of 51 (10 per cent) did the weight exceed 450 Gm., which is the upper limit set by McMichael[6] for enlargement that may result from passive congestion alone. In 26 cases either ascites or other evidence of portal congestion was found, and in 19 of these (73 per cent) the spleen weighed over 150 Gm., but in only 2 (8 per cent) did its weight exceed 450 Gm. Clearly, the degree of splenic enlargement usually found in splenic anemia is not often caused by the portal congestion of idiopathic cirrhosis of the liver, and we must agree with McMichael[6] that some other factors, probably toxic in character, must play a part.

In some instances, the toxin may, as McMichael believes, originate in the liver. The occurrence of gross and progressive splenomegaly in schistosomiasis of the liver and in cirrhosis caused by injection of silica into the portal circulation seems to support this view, but, in splenic anemia, splenomegaly is often well developed before hepatitis is evident.

The modern tendency is to consider the spleen an innocent victim of the malign influences of portal hypertension or hepatitis or of both, but such a concept is quite inadequate to explain the degree of improvement that follows splenectomy for splenic anemia. Patients who survive operation usually show marked improvement. They gain rapidly in weight and strength and are usually able to return to work. The anemia improves, but not to as great a degree as the improved condition of the patient seems to indicate. Ascites, if present, may disappear and not recur for a period of years. Digestive symptoms are usually markedly improved, probably as a result of reduced portal tension, and pain and heaviness are completely relieved. Gastrointestinal hemorrhage, if present before operation, is likely to recur. It seems to me that whether or not the splenic disease is primary, it plays an important part in the production of Banti's syndrome.

The pathologic changes in the spleen are as much a subject of controversy as is the etiology of the disease. The predominant changes are said to be fibrosis, congestion, and degenerative vascular disease, but these processes, either singly or in combination, are incapable of producing the great splenomegaly that is frequently found. Some proliferative changes in the parenchyma of the organ must also occur. The most satisfactory descriptions of the histologic features have been given by McMichael[6] and McNee[9] and it seems to me very illuminating that McMichael states that the pathologic changes which he so painstakingly describes are identical with those described by Banti, whereas McNee, who seems to have studied exactly similar material, says he has never seen the changes reported by Banti and doubts the existence of the disease in the British Isles. It is not surprising that the "common or garden variety" of pathologist is forced to confess that he knows no characteristics or diagnostic pathologic picture by which the splenic anemias can be identified.

One of the unfortunate by-products of the production of this confused and contradictory evidence has been the development of a completely chaotic nomenclature. As early as 1902, Osler[2] selected the term splenic anemia because it "...seems a less objectionable term than splenic pseudo-leukemia, splenic lymphadenoma, splenic cachexia, primitive splenomegaly, or Banti's disease." What

would he have said if he had been able to add to the list, primitive thrombophlebitic splenomegaly, cryptogenic splenomegaly, hepatosplenomegaly, hepatolienal fibrosis, congestive splenomegaly, splenomegalic cirrhosis, and portal hypertensive splenomegaly? Since none of these names is based on established etiology or diagnostic pathologic processes, it seems wiser to preserve the noncommittal term, splenic anemia, to designate a group of diseases from which, as Osler has stated, "successive raids" have removed definite entities in the past and will remove others in the future.

From the standpoint of clinical diagnosis, the situation is even more confusing. In addition to the idiopathic splenic anemias, we find, first, a very large group of diseases due to known causes, such as schistosomiasis, hydatid disease, and syphilitic cirrhosis of the liver, in which the pathologic changes in the spleen and the resultant clinical syndrome are identical with those seen in idiopathic splenic anemia. And, second, another large group, including tumors, cysts, tuberculosis, kala azar, and malaria of the spleen as well as Gaucher's disease, Hodgkin's disease, and aleukemic leukemia may produce the same clinical picture but show definite and diagnostic differences in the splenic pathology. Most of these diseases can be identified by painstaking laboratory investigation of the blood, bone marrow, feces, urine, and material obtained by splenic puncture, but even when specific medication is applicable, as in malaria, syphilis, kala azar, and schistosomiasis, the splenomegaly and anemia may persist in spite of adequate medical treatment.

At first glance, it would seem that the surgeon cannot hope to establish satisfactory therapeutic indications under these bewildering conditions, but actually his problem is not particularly difficult. Splenectomy is definitely indicated for tumors, cysts, and tuberculosis of the spleen, and is of some value in Gaucher's disease. In Hodgkin's disease and in the aleukemic leukemias, it is of no value, irradiation being much more desirable, but biopsy of the lymph nodes or bone marrow will usually establish the diagnosis and prevent useless surgery. In schistosomiasis, the splenomegaly, cirrhosis, and anemia seem never to respond to specific treatment of the primary disease, and in syphilis, malaria, and kala azar they may not do so. Under such circumstances, splenectomy is as definitely indicated and produces as good results as it does in the idiopathic splenic anemias.

Banti's belief that splenectomy brought about a complete cure or arrest of the disease is unjustified. The degree of relief depends to a large extent on the amount of liver damage that is associated with the splenomegaly. At one extreme we find the true Laënnec's cirrhosis with moderate splenomegaly, advanced disease of the liver,

ascites, and marked portal congestion, and at the other extreme we find cases of great splenomegaly without visible evidence of liver damage. In the first group, the prognosis will always be poor, and little, if any, benefit will result from operation. In the latter group, the patient may be, or at least may appear to be, entirely well for many years after operation. It is apparent that the best guide to prognosis is the degree of portal congestion as manifested by the presence of ascites and the occurrence of hemorrhages from the gastrointestinal tract, particularly from the stomach. Whipple and his co-workers report that 7 out of 11 patients who had preoperative hematemesis died of the same complication, and that their mortality in cases of Laënnec's cirrhosis was 22 per cent, compared to 13 per cent in cases showing no demonstrable portal obstruction. Stiven,[10, 11] whose general operative mortality for splenectomy in Egyptian splenomegaly was 13 per cent, lost only 5 per cent of those cases without ascites. Eliason and Johnson[12] lost 6 out of 7 cases who showed evidence of severe liver damage. Some surgeons consider gastric hemorrhage and ascites contraindications to operation, but this seems an extreme view, for such cases are doomed if splenectomy is not done and they may lead useful lives for many years if they survive the operation. Patients without severe liver damage have frequently lived 20 years or more after operation. Rosenthal[13] considers a normal or high platelet count a contraindication to operation because of the danger of postoperative portal thrombosis due to the great increase of platelets which follows splenectomy. This danger certainly exists, but it is by no means certain that the thrombosis is due to the increase in platelets, and most observers are not deeply impressed with the importance of this observation. Portal thrombosis was not recognized by Eliason in 21 cases; it was seen in 2 of 71 cases at the Mayo Clinic,[14] and Whipple and his associates have seen the complication only once in 106 splenectomies, 52 of which were for splenic anemia.

Conclusions

1. Splenic anemia is the oldest and most satisfactorily noncommittal name for the symptom complex of unknown and probably diverse etiology characterized by splenomegaly; secondary anemia; leukopenia and sometimes thrombocytopenia; portal congestion, with or without demonstrable obstruction; cirrhosis of the liver, usually of the portal type; gastric hemorrhages; and ascites. The splenomegaly, anemia, and portal congestion are the only constant features.

2. The pathology of the disease is not agreed upon, but it certainly includes fibrosis and congestion of the spleen with degenera-

tive changes in its blood vessels, both intrinsic and extrinsic. These changes may be brought about by a variety of diseases of known etiology. The mechanism by which they act to produce the splenic pathology is unknown, but they have in common the faculty of causing cirrhosis of the liver.

3. The symptom complex of splenic anemia may be produced by a variety of totally unrelated diseases, most of which, however, respond favorably to splenectomy.

4. Splenectomy is the most useful treatment for the splenic anemias, and both the operative mortality and the ultimate result are good or bad as the liver damage is small or great.

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PELVIC SUPPORTS—THEIR INJURY AND REPAIR

C. J. Andrews, M. D., F. A. C. S.
Norfolk

IN SOME of the lower orders of life the penalty for reproduction is death. This extreme penalty is now seldom exacted of the human species. There is definite evidence that maternal mortality is at long last on the decrease throughout this country. Injuries incident to childbirth are probably also becoming less common as obstetric methods have become more efficient. It may be interesting to note that in a series of 5,700 private gynecologic patients, prolapse, rectocele or cystocele has been the diagnosis recorded in only 163 patients, or about 1 in 35 gynecologic patients had prolapse of some kind. It is probable that this represents a somewhat larger proportion of prolapse than that which actually exists, since 68 per cent of prolapse cases were referred by physicians for operation. A greater percentage of other gynecologic patients came of their own accord. All this number did not come to operation. A few continued to use pessaries though I have not seen one which I have advised to continue with a pessary. I have occasionally made the mistake of using a Gelhorn or doughnut pessary to keep the uterus in until an ulcer was healed, only to have the patient so well satisfied that she was unwilling to be operated upon. Unfortunately, these may return after years when no support will hold and then the patient's health or infirmity may make the problem complicated. Bleeding is the one symptom which often causes these women to seek help. While this is usually due to ulceration, the fear of cancer is a motivating power with some. There are no doubt many cases of prolapse who never apply for treatment. This may be at least partly due to the fact that there is a rather widespread belief among the people and even among practitioners of medicine that the results from operation do not justify the undertaking.

After giving due consideration to its decreasing numbers, childbirth injuries still produce a considerable volume of disability and suffering. Methods are available which give reasonably satisfactory results in a very high percentage of cases, but probably no patient was ever completely structurally cured of a serious injury to the pelvic supports. Obviously the prevention of these injuries is of paramount importance. Though some of these injuries follow normal labor, the vast majority result from difficult or unsuccessful labor. Some consideration of methods which may be expected to prevent the severity and frequency of injuries may be helpful. One answer to this is to be found in prenatal care including planned labor based on findings before labor begins. There can be no doubt that

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cesarean section is resorted to often when it is not advisable but I am sure there are some who should have elective section rather than disastrous labor, to be followed by injury which cannot be fully corrected. This practice does not involve necessarily a high incidence of sections. Many eminent obstetricians hold this view and yet have actually a very low incidence of sections. It does mean an elective section rather than any operative procedure after the patient has been long in labor. No one's information or judgment will ever be so good that a section may not sometime be done unnecessarily but it has been my experience that I have more often regretted not doing a section rather than doing one.

There are certain errors in the conduct of labor which may affect the pelvic supports. Urging the patient to bear down when the cervix is not fully dilated may be harmful, and of more importance is forceps or version before full dilatation. There are a few cases of persistent posterior position in which it is necessary to apply forceps before the head is completely out of the cervix or even before the cervix is completely dilated. Here the head may be rotated by the Scanzoni method and left for further progress by nature or the cervix may be incised. Usually, when ample time has been allowed by analgesia and if the opinion as to disproportion has been correct, the cervix will usually be found to have no resistance after the rotation has been done. Local pudendal nerve block[1] gives excellent relaxation of the perineum and reduces incidence of injury. Ironing the perineum is probably highly injurious to the outer and recto-vaginal supports. Medium episiotomy, if properly sutured, does much to protect the integrity of the pelvic floor. The mediolateral episiotomy, if extended to the side of the rectum, is much more difficult to suture successfully and if not may be followed by poor support. Laceration of the lateral wall of the vaginal tube is not uncommon and if not recognized and properly sutured may be followed by rectocele or cystocele. Where there is any reason to suspect injury to the cervix this should be inspected and sutured if needed.

Mismanagement[2] of the third stage of labor may contribute to injury to the uterine supports. The use of the fundus as a piston to expel the placenta is advised in many textbooks. This is a possible explanation for uterine prolapse following otherwise normal labor. Sub-involution with retrodisplacement, slight injury of the uterine supports and possibly an infected cervix may result in prolapse. Correction of the retrodisplacement, and application of suitable support together with office cautery of cervix may prevent a later prolapse. Even if the support is worn only long enough for involution to become complete the result may be good. There is no intention of suggesting operation for correction of uncomplicated retrodisplacement, but there is a type of retrodisplacement, with or with-

out some minor pelvic disease, associated with cervicitis, first degree prolapse and moderately relaxed perineum in young women who are restored to health by coagulation and conization of the cervix, perineorrhaphy and operative correction of retrodisplacement with suture of uterosacral ligaments. These are simple procedures which may prevent advanced prolapse, particularly after menopause. It leaves the patient in a suitable condition for future labors if desired. In a series of approximately 2,000 private obstetric deliveries which I have personally attended, I am able to find four who later needed operation for prolapse. The first had a labor of 4 hours, 15 minutes duration, the baby weighing 8 lbs., 15 oz., position anterior, episiotomy and perineal forceps used. The second had two children; the first a successful labor and no injury; the second labor was complicated by placenta previa lateralis and was treated by dilating bag followed by version and extraction. The baby weighed only 5 lbs., 5 oz. A 1½ lb. weight was used to control the bleeding. Rectocele, cystocele and prolapse, second degree, resulted. Possibly the dilating bag can be blamed here. The third patient had a perfectly normal labor with episiotomy and perineal forceps; the baby's weight was 7 lbs., 13 oz. After the labor a small cystocele was noticed. The second labor was complicated by a posterior position which required rotation and delivery though the head was low; the baby's weight was 7 lbs., 14 oz. A large cystocele and first degree prolapse resulted. The fourth patient also had two labors; the first an easy labor, left anterior oblique position, baby's weight 7 lbs., 4 oz. Perineal forceps were used following episiotomy and no injury resulted. The second labor required mid-rotation and forceps for an arrested posterior position and delivery was not difficult. Baby weighed 7 lbs., 1 oz. Delivery of the shoulder was noted as being difficult.

Analysis of these cases would suggest that posterior position was a contributing cause in two, a bag in one, and in one case no apparent cause is recognized unless the quick labor and rather large baby could be responsible. This patient was a fat individual with very poor structures and a low metabolic type. These procedures had been done in many and some in almost all of the series of 2,000 deliveries with excellent results.

Prolapse involves such a large number of conditions and associated pathology that a fixed rule for its treatment is not possible. There are certain principles which it is necessary to recognize but each operator will be influenced by his own training, observation and experience in deciding on the method to be used in a given case. Each one will have his favorite method which will more often be used. I acknowledge the obligation which we are under to those who have

developed these standard methods now available. These are continually being changed and developed by individuals in efforts to overcome disadvantages and improve results. The vaginal route is now acknowledged by probably everyone, including myself, to be the most satisfactory way to attack prolapse, particularly of the complete type or procidentia. Any successful operation for prolapse must depend upon a consideration of the pelvic supports. In recent years several very illuminating studies have been made.[3] Mengert evaluated the weight bearing properties of the various structures and showed that the paracervical and upper paravaginal structures furnished most of the support. The structures consist principally of the lateral cardinal or Mackinrodt ligaments, the sacrouterine ligaments posteriorly, and the cervicopubic fascia in front. Curtis[4] has further demonstrated this and described the musculofibrous tubes which surround the bladder, vagina and rectum, and which fusing between form the pubocervical and rectovaginal supports. These are attached below to the perineal fascia and all are fused with the viscera. It is obvious that no intra-abdominal suspension operations can be expected to cure this hernia, and for the same reasons vaginal hysterectomy or any other operation, unless combined with repair of these supporting structures, is not efficient.

During the child bearing period some type or modification of the Manchester operation is available, though the cervix is not amputated if it is possible to avoid it as it may cause a dystocia during the next labor. After the child bearing period some other methods are available. The Watkins interposition operation is popular in some excellent clinics. Its application is limited. The uterus must be normal. It was originally intended for the treatment of cystocele. In cases where no contraindication exists it will relieve the cystocele and prolapse in a large percentage of cases if suitably combined with plastic repair. There are certain disadvantages and risks incident to its use. The danger of developing pathology later, particularly cancer, must be considered. Bladder symptoms sometimes occur and it is surprising that this does not follow oftener. The fundus forms a mass under the base of the bladder much like an enlarged prostate in the male, resulting in residual urine and cystitis, certainly in some cases. Vaginal hysterectomy has many advantages, particularly that of removing the functionless, possibly pathologic uterus. If there is any evidence of bleeding referable to the uterus, this is the method of choice. Even in the hands of the most experienced operators unsatisfactory results sometimes follow. Richardson[5] reports 30 per cent of unsatisfactory results from vaginal hysterectomy. Some of these are probably minor but vaginal prolapse does sometimes occur. This happened in one of my cases; I believe this resulted from unsuccessful suture of the sacrouterine

ligaments and the failure properly to evaluate a beginning enterocele. This complication was treated by the abdominal method of Payne.[6] A Cameron light is passed into the vault of the vagina and by this means the structures are located. The vaginal vault is imbricated and sutured to the broad ligaments. I have treated one other prolapse following vaginal hysterectomy successfully by this method. Richardson, becoming dissatisfied with vaginal hysterectomy, devised his composite operation which is similar to the Manchester-Fothergill except a supravaginal hysterectomy which is done through an anterior cul-de-sac opening. The repair of the supports is accomplished in very much the same way as the Manchester operation. This plan is not suitable if the fundus is too large. It gives full benefit of the parametrial structures and should add another useful method. I have most often used a modification of the so-called Manchester-Donald-Fothergill method for all ages unless the uterus needed to be removed, or when the condition of the patient, either from extreme senility or chronic illness, would not justify even this amount of surgery. So many excellent illustrations of this operation have been published that a repetition would serve no useful purpose.

It might be of interest to mention some of the variations used by others which I have found practical and helpful. As cystocele is so often associated, the treatment of this is really a part of the procedure. After the anterior vaginal wall flaps have been dissected, the pubocervical fascia is separated from the vaginal wall by incising the attenuated edges with the knife and extending the dissection widely to the sides as described by Ward,[7] leaving the fascia attached to the bladder. This avoids hemorrhage from bladder veins. The bladder is separated from the cervix by picking up the ligament which attaches the bladder to the cervix and cutting with scissors, then by alternately opening and advancing the points. Gauze dissection will complete the separation. The cervix is amputated if indicated. The cervix is closed by an inverting stitch similar to the Sturmdorf closure. I have not usually used the Fothergill stitch. If the amputation is very high the lateral ligaments may be separated and sutured together in front of the cervix. The anterior pubic ends of the cervicopubic ligament are sutured together. When the level of the internal os is reached the stitch includes the wall of the uterus at this point, when these sutures are tied the cystocele disappears. Two of these sutures, inserted in body of uterus, are used. The first sutures in the anterior portion of the fascia are particularly useful in controlling relaxed vesical sphincters. This may be modified by the plan of Kennedy or Kelly to control incontinence. Closure of the anterior vaginal wall by sutures which include previous suture line in fascia to prevent dead space completes this part of the operation. The repair of the vaginal floor and perineum is

carried out on a similar plan. The dissection and suture of the rectovaginal fascia is most important, and carried out on the same plan as the repair of the cervicopubic fascia. This structure is not so well defined as the former. In well developed rectocele, it is nonexistent in the midline and dissection must be carried sometimes far laterally to identify the edges. In a large rectocele this hernial opening extends almost to the cervix and must be completely dissected and sutured if a cure is to follow. Suture of the levators or perineal fascia ties in with this. This principle is followed in the primary suture of the perineum, although it is not necessary to dissect the fascia. It is known to exist and though it may be injured it has not been destroyed and may be included in sutures if placed with this thought in mind.

The preoperative preparation and postoperative care is important. All infection and ulcers in the vaginal tract should be cured before operation. If possible, the urine should be sterile. The association of prolapse with general metabolic diseases is well recognized but must be kept in mind. Diabetes and hypothyroidism must often be treated before operation. I have had only one case of wound infection in the vaginal tract. This patient had infected urine and accidentally neglected retention. The cystocele had been large. When the stitches broke down the bladder protruded. Rather long postoperative care was needed to get this again covered with epithelium and clean. The second attempt was successful. Since that time, I have used a retention catheter for several days postoperatively. A plain catheter supported by thin adhesive strips is used. I have followed the plan of placing a light vaginal packing at the end of the operation. This seems to prevent accumulation of serum in the vaginal tract. It is removed in 48 hours and a douche given daily. Dyspareunia has been complained of later by a fair number of these patients; occasionally an adhesion in the vaginal tract may be responsible, but in others the vagina may be slightly tight and the scar tissue sensitive. Elliott treatments have been most successful in relieving this most disappointing accompaniment of an otherwise excellent result. A biopsy is made at the time of operation in all cases. One patient, age 48, with a large cystocele and prolapse to the vulva in which there had been no suspicion of cancer, was reported the next day to have adenocarcinoma. Fortunately, I had placed radium in the fundus to end the periods. This patient was obese and also had diabetes. I left the radium in long enough to give adequate treatment. About three weeks later, the uterus and appendages were removed by the abdominal route. As a very high amputation of the cervix with separation of the cardinal ligaments had been done, the hysterectomy was remarkably easy with the exception of the separation of dense pelvic adhesions which had obvi-

ously followed a previous pelvic inflammation. No trace of residual cancer was found in the uterus after its removal. As this patient has been operated upon only about 18 months, it is too early to call her cured.

I have carefully examined records of 100 consecutive cases of prolapse which have come to operation. Forty-five of these were treated by the Manchester-Fothergill technic alone, 22 by Manchester and laporotomy. In these cases there was some pelvic pathology which seemed to require treatment. Vaginal hysterectomy was done in 6 cases. In all these cases except one the vaginal hysterectomy was selected on account of a pathologic uterus. Perineorrhaphy or repair of rectocele combined with laporotomy was done in 22 cases. These were first degree prolapses with retrodisplacement and relaxed supports but undoubtedly the condition would have progressed later. Incidentally, this type of case gives more definite symptoms of pressure and pelvic discomfort than complete prolapse. Rectocele repair alone was done in 5 cases. Thirty of these patients were over 50 years of age, the oldest was 74. In addition to the two recurrences mentioned, one third degree prolapse with large cystocele had some recurrence of the cystocele from which the patient complained.

With these exceptions, the results were good.

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PREMATURE AGEING ASSOCIATED WITH THYROID DYSFUNCTION

J. K. McGregor, M. B.
McGregor Clinic
Hamilton, Canada

FOR some years, members of our group have been conscious of the premature ageing of patients who are the subject of cystic degeneration in the thyroid, especially when the cyst replaces a pre-existing fetal adenoma. We have postulated that the contents of these cysts have been absorbed into the general circulation, producing toxic symptoms entirely different from those caused by hyperthyroidism. We believe that our deductions, based on careful study and correlation of the pathologic and clinical pictures, are fully justified.

When a thyroid cyst developing on a fetal adenoma is studied, we find it almost completely excluded from the rest of the gland. The capsule is thickened as a result of the degenerative process. The connective tissue of the adenoma has disappeared. The cells, if any remain, are flattened out from pressure, and show neither the tendency nor the ability to proliferate.

It is an interesting observation that quantitative chemical analysis of the cyst contents reveals the presence of cholesterol in relatively large quantities.

Clinical study of the patient reveals a history of a "let-down" feeling, a feeling of inward nervousness. Examination reveals the presence of a cystic thyroid, usually with a single cyst. Trophic changes predominate. The patient looks tired and appears to be ten or fifteen years older than the stated age. The skin is markedly changed. It is dry, wrinkled, and lifeless in appearance. The hair is turning gray prematurely, and falls out easily. The nails chip. In short, these are the superficial signs of body ageing. As to sex, females predominate. The fourth decade produces the majority of cases.

It is noted that there is no acceleration of pulse, no exophthalmos, no tremor, no heat intolerance, no loss of weight, no cardiovascular stimulation, and no elevation of the basal metabolic rate; in fact, none of the symptoms of hyperthyroidism.

It will be said that what we observe is merely a picture of the results of a progressive life history of a fetal adenoma. Our clinical experience does not bear this out. We believe that such patients form a distinct group, in which symptoms of toxicity are present,

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and easily recognizable. The only term we have for this is "ageing." It must be clearly understood that this does not mean that the patient, who is suffering from hyperthyroidism caused by nodular toxic goiter, does not show also signs of ageing, as the disease progresses.

We believe that these ideas parallel those of Hertzler,[1] when he says:

> That the thyroid grows us up to adult life is an established fact, and that, seeing its mistake after having fostered our development, it at once proceeds to get us out of the mess it got us into. Why and how we grow old has not been satisfactorily explained. My suspicion is that the thyroid gland, by throwing its gears in reverse, sees to our orderly demise, unless some intercurrent disease or mechanical contrivance anticipates its design.

Most other authors entirely disregard any toxicity of the thyroid not due to hyperthyroidism.

The extent to which cholesterol is a factor in vascular decay is debatable, but physiologists and pathologists who have been consulted, have admitted the possibility of the part it might play in the picture we have just described, having noticed its presence in quantities as a result of degeneration of adenomatous tissue.

The following case, which we observed about twenty years ago, will be illustrative:

> A young lady, 30 years of age, working on our staff, had a palpable adenoma which we advised her to leave alone until it bothered her. She left our staff, and a year later was sent to us as a patient, and the change was remarkable. She looked at least 45, the adenoma had become cystic, she felt let down, her hair had become prematurely gray, her skin was rough, wrinkled, and without life, and she complained of inward nervousness. Her basal metabolic rate was normal, and she had none of the symptoms of hyperthyroidism.
>
> A solitary cyst was enucleated from the thyroid gland. There were no adenomas palpable in the remaining structure. Within six months she had regained her appearance of the year before, but she still had her gray hair. The patient was followed for several years. The grayness of her hair did not increase. She did not show any more signs of body decay, nor did she have any recurrence of her symptoms.

Since this time, we have observed many similar cases. We find they run true to form with little variation. They show the following characteristics in addition to what has already been described:

1. Iodine has no effect upon the symptoms, nor upon the feeling of the patient.
2. The patient never shows any reaction, such as a crisis following surgery.
3. The results of enucleation of the cyst are invariably satisfactory to the patient, and to us.
4. Almost all the symptoms disappear after enucleation, and the incidence

of recurrence is no greater than one would expect in an individual who had never had any dysfunction of the gland.

One particularly interesting observation has been made in our Clinic. In the follow-up, these patients express their reaction to their treatment without coaching on our part, in such terms as, "I feel ten years younger," or "a weight has been lifted." This voluntary statement is made by at least 80 per cent of our patients in this group.

Treatment

On account of its simplicity, no other treatment but surgery has been advised. The operation is a minor one, without risk, and without any reaction. Local anesthesia is ideal. Enucleation of the cyst, and palpation of the remaining gland, is all that is necessary. Seldom is a lobectomy required. Convalescence is short, and the scars are practically unnoticed.

Conclusions

While we do not at this time hope to convince others, as we ourselves are convinced, that there is a toxicity due to the thyroid dysfunction without hyperthyroidism, yet we believe that many patients might have the advantage of surgery, if these early signs, outlined above, were recognized. It makes little practical difference whether the results are due to the absorption of something present in the cyst contents, or to some other toxic agent associated with the thyroid. The fact remains that too much stress has been laid on the basal metabolic rate, and the cardinal symptoms of hyperthyroidism, in assessing the responsibility of the thyroid gland, in whole or in part, for certain phenomena, which we, with increasing frequency, observe in patients. We believe that the cystic thyroid, even in the absence of raised basal metabolic rate and of other evidences of hyperthyroidism, must be held responsible for conditions herewith described.

Note—For the last six years, we have introduced into animals the contents of all cysts removed, and have noted certain trophic changes in these animals. There are no conclusions because these experiments have been done without control, and the contents of other cysts of the body have not been introduced for comparison. We are planning to do this during the present year, and hope to make a report upon our findings at some future date.

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THE STRUCTURE OF A KNEE JOINT IN HEREDITARY ARTHRODYSPLASIA

JOHN H. VENABLE, M.D.

Assistant Professor of Anatomy, Emory University School of Medicine

Atlanta

HEREDITARY arthrodysplasia associated with dystrophy of the nails has been reported by several clinicians. Turner[1], in investigating the family history of two patients, found 35 out of 79 persons in two families affected with arthrodysplasia and dystrophy of the nails, and commented on the remarkable hereditary association of the two conditions. Sever[2] recently presented similar findings in a 65-year-old painter who had been admitted to the hospital because of symptoms later shown to be due to inoperable carcinoma of the pancreas. Upon the death of this patient in January, 1939, the right knee joint (the knee joints were the sites of greatest deviation from the normal) was procured by Dr. Sever for the Warren Anatomical Museum. Dissection of this joint by the author, at the request of Dr. M. M. Canavan, Curator of the Museum, makes it possible not only to describe the joint but to correlate the anatomic findings with the clinical picture described by Sever.

Sever's description of the knees reads:

"Examination showed that there were no patellas where one would expect to find them. The knees were broad. The internal condyle was more prominent than usual, and the intracondylar notch could be easily palpated. The patellas were small and were located on the external aspect of both knees. The quadriceps tendon was small, and the muscle no longer than a finger-breadth. The motions in the knees were practically normal, actively and passively, but active extension was much diminished in power. . . His legs had bothered him in walking, and he had had difficulty in walking fast or going up and down stairs, owing to his weak and mechanically inefficient quadriceps. His work compelled him to stand all day."

In another part of the history it is said,

"On his mother's side, a niece and his uncle . . . had rudimentary nails on all fingers. He knew nothing about their knees."

Turner's description of the knee joints of two patients parallels this description so closely that it could be substituted here without loss of essential details, i. e., lateral position of patellas, prominence of internal condyles, and weakness of active extension.

A roentgenogram of the knees, published by Sever and herewith reproduced in part as fig. 1, shows both patellas articulating with

From the Department of Anatomy, Harvard Medical School, Boston. This work was done while the author was on a Commonwealth Fund Fellowship.

the lateral condyles of the respective femurs where there is a raised articular facet developed in relation with the femoral facet of the patella. The internal condyles are large and a suggestion of lateral rotation of the tibias is evident. The space between the medial femoral and tibial condyles is apparently increased, indicating such separation that it is doubtful if the medial half of the joints took much part in weight bearing.

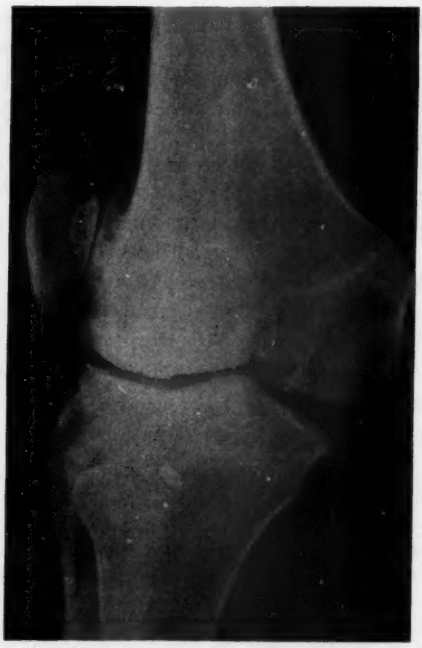

FIG. 1. Roentgenogram of the right knee joint. The lateral position of the patella with the related femoral facet is clearly visible. Note the size of the medial femoral condyles and the lateral rotation of the tibia and fibula. The shadow cast by a calcified mass, located between the popliteus and soleus muscles, is seen superimposed on the upper end of the tibia.

(The author is indebted to Dr. Sever for the loan of the film and to both Dr. Sever and the New England Journal of Medicine for permission to reproduce the illustration.)

The post mortem specimen, consisting of the right knee joint together with the distal third of the thigh and the proximal third of the leg, conforms to Sever's pre-mortem description. It should be added, however, that the tuberosity of the tibia is located somewhat lateral to its normal position, a relationship which indicates lateral rotation of the leg.

Dissection of the structures covering the joint reveals several points of interest. The tendon of the quadriceps femoris muscle crosses the anterior aspect of the knee joint as a wide, thin, tendinous band and inserts into the front of the medial condyle of the tibia, into the tuberosity, and into a restricted area of the lateral condyle immediately adjacent to the tuberosity (fig. 2). The tendon is thickened at its lateral edge, which attaches directly to the tibial tuberosity and contains the laterally placed patella.

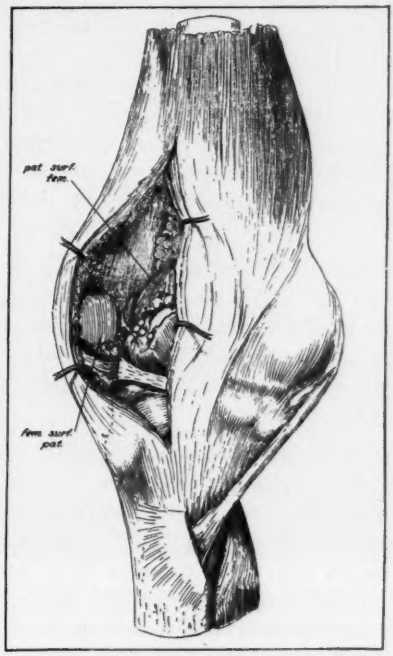

FIG. 2. Drawing of the opened right knee joint. The quadriceps muscle is seen crossing the front of the joint. This has been split vertically between the two parts of the tendon. The femoral surface of the patella *(fem. surf. pat.)* is seen to the left. The opposite femoral facet *(pat. surf. fem.)* is also clearly visible. Behind and above these surfaces the interior of the enlarged laterally displaced suprapatellar recess of the joint capsule is shown.

This portion of the tendon is probably all that could be palpated pre-mortem, a circumstance which explains the clinical finding of a quadriceps tendon "no larger than a fingerbreadth." The capsule of the suprapatellar recess of the knee joint is large and thickened. It appears to have been displaced laterally where it extends well under the iliotibial tract to lie in extensive relationship with the lateral intermuscular septum. All other external features of the joint are within normal limits except for an unexplained

small, solid, calcified nodule between the soleus and popliteus muscles which is attached to the capsule. The shadow cast by this nodule is visible in fig. 1.

The interior of the joint presents a picture of extensive degenerative joint disease. The capsule is markedly thickened and the synovial space contains several cubic centimeters of yellowish, viscous fluid. There are no free bodies (joint mice) present. The patella is small. Its femoral surface is elevated and extends over only a part of the posterior aspect of the bone (fig. 1). The articular cartilage covering the femoral surface of the patella is intact and forms a smooth surface. On the contrary, the patellar facet, which has developed on the lateral condyle of the femur, consists of an aggregation of hypertrophic nodules which have fused into a comparatively smooth facet at the summit (fig. 2). This facet is elongated in a postero-inferior direction corresponding to the arc traversed by the patella in flexion.

There is marginal proliferation of articular cartilage and marginal lipping, both of which are much more prominent on the femur than on the tibia and more extensive anteriorly than posteriorly (fig. 3). In addition, there is a tremendous hypertrophy of synovial villi most marked anteriorly and in the neighborhood of the patella (fig. 2).

The articular cartilage of the lateral femoral condyle is denuded to such an extent that the underlying bone is completely uncovered in some areas. This gives rise to the moth-eaten appearance presented by the lateral condyle in fig. 3. The opposing lateral condyle of the tibia presents the same changes in a lesser degree. The cartilage of the medial femoral and tibial condyles presents a few superficial linear depressions which are quite different from the denuded areas of the lateral condyles (fig. 3).

The anterior cruciate ligament is completely absent. No trace of any part of this intra-articular ligament is demonstrable, as may be noted in fig. 3 which shows the attachments and course of the posterior cruciate ligament. All other ligaments of the joint are within normal limits.

Speculation concerning the essential etiologic factors of this condition must take note of the complete and presumably congenital absence of the anterior cruciate ligament. In normal joints, lateral rotation of the tibia on the femur causes immediate tightening of the cruciate ligaments (a winding of one about the other) which are generally believed to limit lateral rotation. In the absence of the anterior ligament the winding effect would be lost so that the

posterior ligament would serve only partially to immobilize the lateral tibial condyle, while the medial slides forward. Consequently, more than the usual amount of lateral rotation would be allowed. In these circumstances it would be logical to presume an increased tendency to lateral dislocation of the patella.

FIG. 3. Interior of the right knee joint, from in front, after the knee has been flexed to an acute angle to bring the femoral condyles into view. One is looking at the opposing surfaces of both femoral and tibial condyles. Note the absence of cartilage on the lateral femoral condyle (left) and the unusual markings on the medial condylar cartilage. Note also the signs of degenerative joint disease. The intercondylar notch is narrowed and its limiting edges are sharp. The femoral end of the posterior cruciate ligament can be seen at its attachment to the lateral surface of the medial condyle. The anterior cruciate ligament is entirely absent. Part of the medial semilunar cartilage can be seen just below the medial condyle.

Bennett and Bauer[3] have shown that degenerative disease of the knee joint develops in rabbits if the patella is dislocated and held in an abnormal position by sutures. Moreover, they found that a new femoral articular surface is formed in relation to the abnormally placed patella. The degenerative joint disease produced by their experimental procedure is similar to that found in the present dissection.

The most logical explanation for the condition here reported would seem to lie in a congenitally absent anterior cruciate liga-

ment which led to lateral dislocation of the patella. This dislocation, unreduced, served as the basis for other joint changes.

Why such a sequence of events, involving a primary mesodermal defect, should be hereditarily associated with dystrophy of the nails (ectodermal) is unexplained.

Summary

An anatomic dissection of a dysplastic knee is described to permit correlation with a clinical description of the same patient published by Sever in 1938. The chief anatomic defects are the complete absence of an anterior cruciate ligament and lateral dislocation of the patella. Extensive degenerative joint disease is present and is assumed to have developed as a consequence of the primary anatomic defects.

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A CONSIDERATION OF THE TERMINOLOGY AND THE MANAGEMENT OF ANAL FISTULAS

LOUIS A. BUIE, M. D.
Section on Proctology, The Mayo Clinic
Rochester, Minnesota

IT hardly seems necessary to define a term which is applied to so prevalent a disease as fistula in ano. However, inasmuch as it is probable that the difficulties which arise in the management of this disease are largely due to a faulty conception of the meaning of the terms which are used to designate it, an attempt to clarify the problem seems warranted. The confusion which exists may be illustrated by the frequency with which the terms fistula in ano and "rectal fistula" are used interchangeably. The two expressions describe conditions which are not the same and they should not be used synonymously. By the term "fistula in ano" is meant a fistulous tract which originates in the anus and which connects it either with the external surface of the body or with one or more of the neighboring viscera. There must always be at least two openings, one at the source of the fistula and the other at its termination. Manifestly, with a proper knowledge of the true meaning of the word fistula, it will be understood that neither an incomplete internal nor an incomplete external fistula can exist.

It is customary to designate the source or the point of origin of an anal fistula as the internal opening. Only a moment's consideration is required to realize that it is also possible for another internal opening to develop higher in the bowel at a point where an abscess has involved the rectal wall and ruptured through to bring about the terminal feature of the fistulous process. With such a condition, the "internal opening" therefore becomes ambiguous and no idea can be conveyed as to the true meaning which is intended. If, then, we consider the external opening, we find ourselves again in a state of confusion which occurs because this term in its usual acceptance refers to the terminal point of the fistulous process and although an external opening always applies to such a point, nevertheless, the terminal point of the fistula is not always situated externally as, for example, in those instances in which the final rupture occurs higher in the bowel wall or within the cavity of neighboring pelvic viscera.

In order to clarify our position with regard to this system of terminology, it would seem profitable to discard the old terms and to substitute in their place the word "primary" to designate the point of origin of anal fistulas and the term "secondary" to indicate the point at which the fistula terminates. Thus, we will find that

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although the "primary" opening is always internal, we need not become confused if there is another opening higher in the bowel or lower within the anus because we know that these subsidiary openings are "secondary." The "secondary" opening also may occur either on the skin surface or within the wall of the rectum, vagina or bladder without causing concern as to whether there is an internal or external opening.

Etiology

Whence comes the infection to which the onset of the disease is due and through what portal of entry does it gain admission to those structures which finally break down into an abscess and ultimately suffer the development of a fistulous tract? What is the nature of the infection?

Any of the pyogenic organisms which inhabit the colon are capable, on gaining admission to normal tissues, of producing inflammatory changes which may terminate with the formation of an abscess. Tuberculosis may be associated with anal fistulas and we should not allow ourselves to be shunted off from the proper management of the disease because we find that the anal lesion is complicated by tuberculous disease either locally or in some other part of the body. Nowadays, when anesthesia may be administered satisfactorily and with no risk, the fistula should be treated properly not only because of the troublesome nature of the local disease but because of aid that the treatment will afford the patient in overcoming the pulmonary disease.

Pathology

Any accident which breaks tissues which invest or are adjacent to the anal crypts may terminate in the development of anal fistula. Accompanying such a break, micro-organisms capable of producing inflammatory changes are admitted to subcutaneous and submucous tissues. Here, within the anal crypts, we find the primary openings of anal fistulas. In most instances primary openings will be found to occur singularly although it is possible for several openings to exist. At such times it will be found that the crypts are open and that they are connected with each other by submucosal or subcutaneous tracts. The posterior half of the anus is most frequently involved.

The first stage in the development of an anal fistula begins with the involvement of the anal crypt. From that point the fistula extends into the adjacent structures and enters the second phase, which may best be described as the stage of burrowing. The disease of the crypt is characterized by inflammatory changes such as edema of the papillae and of the margins of the crypt, redness, purulent discharge and bleeding.

During the second stage the infection extends in various directions from the primary opening. The course may be: (1) immediately beneath the skin of the anal margin, (2) through the body of the external sphincter, (3) between the anal sphincters, or (4) superficial to or directly through the internal sphincter or the circular muscle fibers of the lower part of the rectum.

In the third stage an abscess forms and it is commonly known as an "ischiorectal" abscess. The term "ischio-anal" is often more nearly correct. Sometimes the abscess involves the tissues of the supralevator spaces. Then such terms as: (a) "right pelvirectal" or (b) "left pelvirectal" should be used. Other abscesses such as retrorectal, right and left ischio-anal, perirectal, perianal, pararectal and para-anal may occur.

When the burrowing sinus passes through or in the vicinity of the anal sphincters, the inflammatory process produces a spastic reaction on the part of the constricting muscle bands and the spastic muscles usually constrict the walls of the sinus and prevent evacuation of the purulent discharge in that direction. Therefore, the infection is confined to the loose areolar tissues adjacent to the anus and rectum.

Thus we come to the fourth stage in which the abscess progresses until the cavity becomes filled with pus and finally ruptures or a surgical operation is performed to evacuate the contents of the abscess cavity.

Symptoms

The abscess usually provokes the earliest subjective evidence of the disease. Then pain is the chief symptom and swelling the most important sign. A small abscess within the grip of the external anal sphincter produces excessive pain; one which is much larger and situated in the ischio-anal space, away from the anal musculature, will not produce so much discomfort until it has reached extensive proportions. Visible swelling is sometimes absent in cases in which deeper abscesses form, and patients so afflicted often complain of pain long before the abscess is discovered. Fever and prostration are often present before the abscess is drained.

Following the evacuation of the abscess, there usually remains a persistent draining sinus. In the simple forms of the fistulas the sinus may close temporarily.

Diagnosis

The appearance of a discharging sinus in the tissues adjacent to the anus of a patient who gives a history of an abscess which has ruptured or which has been incised is almost conclusive evidence of an anal fistula.

The solution of the problem, whether during the pre-fistula stage or after the fistulous tract has formed, is often determined by the anoscopic examination, during which the character of the anal crypts may be carefully investigated. Often there will be evidence of disease in the anal crypts and this is usually more evident after the fistula has developed than during the stage of abscess. When the anal margins are separated, a spot or even a small stream of pus may be observed to come from behind the pectinate line and sometimes a definite opening may be discovered. At other times only a tuft of granulation tissue or a scarred region within the crypts may be discovered.

All probing of sinus openings, both primary and secondary, should be left until anesthesia has been instituted, and if injections of colored solutions, bismuth and lipiodol must be made, they should also be deferred until sensation has been abolished in the region to be examined.

The patient is scarcely ever aware of any disorder of function prior to the appearance of an abscess. Doubtless, we see lesions of the anus which begin as do anal fistulas, but instead of progressing to the development of a sinus, they produce other lesions such as cryptitis, papillitis and ulcer.

Treatment of an Abscess

In general, it is probably better to allow the abscess to approach as nearly as possible the point of rupture before operation. By this plan, the abscess wall becomes well outlined and is allowed an opportunity to approach nearer to the point where incision may be made or rupture may occur. In the case of a deep abscess, incision must often be made through several centimeters of healthy tissue and such management may result in a stormy convalescence because there is such a large region through which the purulent discharges from the abscess may be absorbed. After incision or rupture of an abscess, the pyogenic wall contracts into a tube-like fistula. Those abscesses which involve the ischio-anal space usually point somewhere in the surface of the perineum, the anococcygeal region or in the skin of the buttocks. Such an abscess should be opened by a cruciform incision and the points intervening between the cross incisions should be cut away in order to leave a roughly circular opening. This opening should be spacious enough to make any interference with the evacuation of the pus impossible.

Rarely, the ischiorectal abscess is connected by an isthmus with another portion of the abscess situated above the levator ani muscle. Such a condition may be suspected when a greater volume of pus is expelled from the abscess than is possible for the abscess to accom-

modate if it is limited to the ischiorectal space. When the abscess is first opened, one is not justified in exploring the cavity very thoroughly in order to solve this question. In the acute stage, the pyogenic wall of the abscess is soft and friable, and carelessness, even during digital examination of the cavity, may cause one to break through this wall with serious consequences. Septicemia and pyemia are not unknown following such errors of judgment.

When the pelvirectal or retrorectal spaces become involved but there is no fluctuation externally, and when the abscess is discovered adjacent to the upper walls of the rectum, it may become necessary to make an incision through the rectal wall.

The duration of time which should elapse between the drainage of the abscess and the performance of a fistulectomy varies within wide limits. It is desirable for the acute condition to subside until the abscess cavity has been reduced to a fistulous tube. This is not always possible but the walls of the cavity may become fibrous and their outline more definitely limited and the sacrifice of tissue which usually accompanies a fistulectomy may be reduced. An elapse of only a few weeks is usually sufficient but with large abscesses it may be advisable to wait several months.

Treatment of the Fistula

The chief factors concerned in anal fistulectomy are four in number: (1) find the primary opening, (2) trace the fistulous tract or tracts, (3) cut away those structures which are external to the primary opening and by incision convert the fistulous tunnels into open ditches, (4) treat the cavity so that it will heal from within outward without the development of other fistulous tracts.

If possible, the operation should be begun at the source of the disease and the fistulous tract should be opened from the primary opening toward the secondary opening. When a probe is forced in through the secondary opening, it is liable to perforate the wall of the fistula and give a false conception of its course. Such an accident may misdirect the efforts of the surgeon during the operation, especially if he proceeds to cut blindly along a grooved director. Such a maneuver may pierce the rectal wall and create the impression that the source of the disease is high. Whether one begins the operation at the primary or at the secondary opening, he should insert the probe only a short distance at a time before splitting the fistulous tube.

The anus should not be stretched forcibly before exploration of the tract is begun. If the fistula is complicated by a stricture of the anus, it may interfere with one's effort to expose the interior of the anal canal, and it often is a great temptation to dilate the outlet

thoroughly in order to provide proper exposure. If possible, one should avoid breaking the contraction in an effort to expose the anal crypts. Usually the fistula passes through the fibrous deformity surrounding the anus and forceful dilatation is likely to break the tract. Then, the problem of ferreting out the course of the fistulous tube may be difficult, if not impossible.

When searching for the primary opening, one's efforts are often rewarded by an immediate discovery of the point of invasion of the infective organisms. Usually one finds an open sinus varying from a millimeter to as much as a centimeter or more in diameter, and often pus will be seen as it escapes into the anal canal. Success in discovering the obscure primary opening depends a great deal on one's ability to recognize pathologic change in the crypts. In the absence of a visible opening, one should seek other evidence of disease. Pus may appear behind the pectinate line or pressure on the overlying fistulous tract may produce a spot of pus in a crypt. A tuft of granulation tissue may be the only sign of disease. Sometimes there may be a scarred region within the crypt, and by grasping the margin of the secondary opening with a clamp, one may observe a definite tug at the site of the scarred crypt.

None of these discoveries may be made and the surgeon may note only that one or more of the anal crypts is deeper than usual and shows evidence of inflammation. A papilla or several papillae may be hypertrophied, edematous or fibrous.

The operation is begun by bending the probe so that it will conform to the curve of the fistulous tract, and by inserting it into the primary opening it is brought along the tract of the fistula. An incision is then made through the tissues superficial to the probe until the entire fistulous tract is laid open and the probe lies free in the wound. It is not always possible to pass the probe through the entire course of the tract; it then becomes necessary to insert it for a distance, lay the tract open that far, then reinsert the probe further and continue the incision in that manner until the entire tract is opened. At other times, owing to the contraction of the anus, it may not be possible to insert the probe through the primary opening at all. It then becomes necessary to explore the tract from the secondary opening toward the anus. As the tract is laid open in that direction, undue pressure on the probe should be avoided. By scrubbing the walls of the fistulous tract with dry gauze, the point where the fistula turns toward the anus can be discovered. Usually, the tract is filled with grayish, gelatinous débris. After this has been wiped away, there will be found one point at which this grayish deposit cannot be removed. The probe usually will find its way into a portion of the remainder of the tract when passed through this

deposit. By repeating this procedure the incision of the tract is carried to the primary opening. In the same manner, subsidiary tracts are discovered and incised. When the main tract is opened to a point adjacent to the anal wall, it is not difficult to establish the connection of the tract with the primary opening. Then the probe is passed into the anal canal through the primary opening and the structures external to the tract are divided.

All fistulas are not so simple in structure. More complex varieties present greater difficulty but the problem in each type of complicated fistula is essentially the same. Following a high abscess, fistulous tracts may course from the primary opening upward to the abscess cavity and then may angulate and continue downward toward the external surface of the buttocks and perineum to the secondary opening. In such a case it is advisable to explore the tract through both the primary and the secondary openings. It may be possible to insert one probe through the primary opening and direct it upward into the pelvis until it reaches the abscess cavity. Then when one inserts another probe into the secondary opening, it may pass through the second limb of the fistula toward the same cavity where the two limbs of the fistula meet. Then, by manipulating the two probes they may be felt to touch each other. The operation is begun by cutting through the anal wall at the site of the primary opening. Then, after making an incision from this wound to the secondary opening, the incision is carried deeper and deeper until both probes lie free in a deep, open wound with the two limbs of the fistula laid open as they come together at the apex where the abscess cavity exists.

Operations on the simpler types of fistulas are completed by dissecting the overhanging edges of the tracts and the tissues of the anal margin in such a way as to facilitate the application of dressings during the postoperative period. When tracts are superficial they may be excised. When they are very deep the walls of the tract cannot be excised without excessive destruction of tissue but usually they will heal satisfactorily after they have been scarified with a curet.

Occasionally, from a simple fistula, a subsidiary tract may run upward into the pelvis and end blindly in that region. It is not necessary to lay such a tract open or attempt to remove it by dissection. The source of the infection responsible for this fistulous limb is in the primary opening, and if this has been uncovered, the infectious discharges from the rectum can no longer reach this branch of the tract. If left alone, the subsidiary tract will shrink down into an inactive fibrous cord.

If such a tract has broken through the rectal wall, the condition requires a more elaborate operation. Then an incision must be

carried through all of those tissues which intervene between the subsidiary tract and the rectal wall, all the way to the high secondary opening. The rectal wall itself is split through the site of the primary opening and the subsidiary tract is laid open in continuity with the rectum. This operation is partly an intrarectal operation, and unless this procedure is followed a cure may not be expected.

The complete internal fistula presents another problem. From the usual primary opening the tract courses upward into the pelvis and the secondary opening appears in the rectal wall. The operation for such a fistula must be performed intrarectally and the fistulous tract exposed in the same manner as the external types of fistulas. When a large chronic abscess cavity is associated with such a fistula, it may be necessary to cut the anal sphincter in order to drain the cavity properly.

Fistulas which involve the bladder, vagina and other pelvic organs fortunately are rare. The problem presented by each usually is determined by the nature of the complication and therefore no treatment can be described which will be suitable for the relief of all. The tracts of some anal fistulas are circular; the tracts of others are extensive and unusual in distribution. Others are of the familiar "horseshoe" type.

There is no reason why the sphincter should not be cut through in more than one place when there are two independent sources of the disease, and the old idea that the sphincter muscle should not be cut in an oblique direction may be disregarded. An old rule, but still a good one, is this: never suture a wound following fistulectomy except when the tracts run superficially and far out into tissue adjacent to the anus. Then, the distal limbs of the tract may be sutured but the wound should not be closed near enough to the anus to permit the rectal discharges to accumulate beneath the suture line.

By following the general principles which have been laid down, the surgeon will find that most anal fistulas will respond to surgical treatment. During the performance of the operation one should arrange the tissues so that they will heal from the deepest portions of the wound toward the exterior, wall against wall. Then, with proper care, the healing process may draw the structures together and form a block of scar tissue. Thus, the remains of the anal sphincter will become attached to this scar and as the contractile power is exerted the anal orifice will close in the same manner and with the same degree of efficiency as before the operation. This desirable type of healing will not take place if packing is placed in the wound immediately after operation and is allowed to remain there for a number of days.

Usually incontinence is not due to the surgical destruction of muscle but to the fact that the cut ends of the muscle fibers have not been drawn together by the proper kind of scar. The postoperative management of anal fistulas is the same as that which should be provided for any type of infected wound. Suitable irrigations should be given often enough to keep the wound clean. The rectum should be kept clear of feces so that discharges will not constantly bathe the wound surfaces. If it is felt that the bowel should not act for a number of days, this can be accomplished by providing a nonresidue diet which, if properly worked out, will obviate the use of sedatives like camphorated tincture of opium and lead and opium pills. Hot wet applications are of great value in relieving discomfort and controlling the inflammation. The wound should be kept clean at all times. As soon as it is soiled by fecal discharges or by the accumulation of purulent discharges on the dressings, cleansing measures should be instituted and new dressings should be applied. This is important even if dressings must be changed several times a day. Postoperative management should be considered of equal importance with the operation.

PROSTATIC DISEASE

With Special Reference to the Various Causes and Types as Well as Their Treatment

ROY B. HENLINE, M. D., F. A. C. S.
New York

IT has become so common for all of us to refer to the various types of prostatic surgery as "removal of the prostate" that many surgeons actually forget that the prostate is rarely removed. The hyperplastic tissue, or so-called adenoma, only is removed, and the functioning prostatic glands, or their remnants, still remain firmly attached to the capsule. Removal of this hyperplastic tissue is frequently all that is required to establish adequate bladder drainage, and the remaining uninvolved prostatic tissue may reasonably be left behind. It is not uncommon, however, for the remaining prostatic glands to be the seat of a pathologic process, requiring surgical removal.

The literature is filled with discussions relating to the advisability of removing obstructing hyperplastic prostatic tissue by the suprapubic, perineal, or transurethral methods. Each of these methods of approach has its advantages and should be used in certain patients. However, it is our opinion that the nature of the prostatic disease in a given case should first be determined as nearly as possible before the type of surgery is decided upon. A study of the pathology of the prostate reveals the frequency with which certain operative procedures fail to remove the entire source of the symptoms. Before deciding on the best surgical approach in any patient, we should determine the particular procedure which will relieve the patient's symptoms with the least morbidity and mortality and offer him the best future outlook.

HYPERPLASIA (ADENOMA) OF THE PROSTATE

Excepting infection, most prostatic enlargements are probably incited by an imbalance of male and female hormones in favor of the female hormone, which occurs near the age of 50 years. There is, as yet, no pathologic evidence that hormonal therapy will inhibit or stop this process once it has begun.

Deming points out that so-called adenomas of the prostate are, in reality, a hyperplasia of the prostatic ducts stimulated by a solid fibromuscular mass in the muscular wall of the prostatic urethra. These masses develop centrally just underneath the mucous membrane of the prostatic urethra.

From the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital—Cornell Medical Center.

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.

These fibromuscular masses, which incite prostatic hyperplasia, are not found in the lateral lobes, posterior lobe, or the capsule of the prostate. They develop only in the muscular layer directly underneath the urethra and stimulate prostatic hyperplasia, not from the existing prostatic glands, but from the ducts of these glands just before they open into the prostatic urethra. As these areas of hyperplasia increase in size, they compress the true lateral and posterior lobes toward the capsule and assume the position of these lateral lobes on each side of the urethra. According to Deming, this hyperplasia is not just an enlargement of the existing elements but probably is a neoplastic duplication by many times. As these lateral lobes increase in size, the prostatic urethra is distorted with an anteroposterior elongation (fig. 1). Since the *ducts* of the prostatic glands are involved in this hyperplasia, the drainage from these glands is gradually obstructed. The functioning prostatic glands are gradually compressed toward the capsule by these enlarging hyperplastic areas until they finally may be almost obliterated. Between this hyperplastic tissue (adenoma) and the compressed prostatic glands, a line of demarcation is present which permits the adenoma to be easily separated and enucleated. In many instances the compressed prostatic tissue and capsule may be left intact without causing any future disturbance. This is by far the most common cause of prostatic obstruction seen in advancing years.

The removal of obstructing hyperplastic prostatic tissue may be adequately accomplished by the suprapubic, perineal, or transurethral method by many urologists. When such an uncomplicated hyperplasia produces urinary symptoms of obstruction, most urologists first determine the size, location and extent of the enlargement and, considering their surgical skill, decide the best surgical approach for them to use in removing the obstructing tissue. There are many other factors, such as retention, renal function, general condition, which also influence us all in determining the best method of approach in a specific case. Since these have been thoroughly discussed in the literature, I shall not repeat them here.

Other Prostatic Pathology (Infection: Calculus)

Surgery of the prostate is often indicated for conditions other than simple hyperplasia (adenoma). Prostatic infection, calculi, and malignancy may coexist with hyperplasia. In patients with such complications, one should choose the surgical procedure which will relieve not only the urinary obstruction but any other existing disease of the prostate.

The entire prostate undergoes a change at about 50 years of age. Moore has shown how poor drainage from the prostatic glands

predisposes to the formation of a nucleus of desquamated epithelium and debris to form corpora amylacea. These foreign bodies are formed in the functioning prostatic glands in contradistinction to the hyperplasia which develops near the urethra from the gland ducts. These corpora amylacea are frequently surrounded by round cell infiltration and fibrosis in the prostatic gland itself. Calcium salts may be deposited around these nuclei to form prostatic calculi.

These corpora amylacea, or prostatic calculi, often surrounded by evidence of infection, may be present for years, without causing any symptoms. Sometimes, however, a marked infection develops and symptoms are produced that require surgical intervention. In other patients hyperplasia (adenoma) may develop in this same prostate in the region of the ducts near the urethra. As this

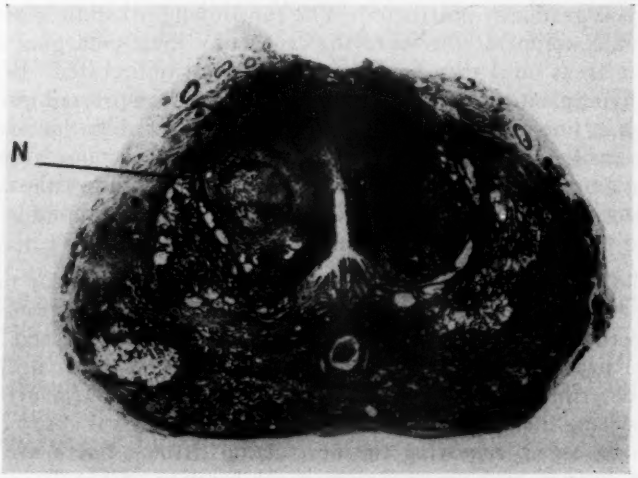

Fig. 1. Photomicrograph x 2. Cross section of prostate of man, aged 47 years, showing nodule "N," one-half of which is glandular and the other half aglandular. Note fibromuscular masses on each side of urethra invaded by glandular hyperplasia. True prostatic glands are being compressed toward the capsule. Note beginning anteroposterior elongation of urethra. (From Deming.)

increases in size, the corpora amylacea, infection and calculi, if present, are compressed toward the capsule with the prostatic glands; the ducts from the prostatic glands are gradually occluded and the infected prostatic remnants may no longer drain into the urethra. Some glands become atrophic, while in others infection or calculi may be walled off by the enlarging hyperplasia.

In the latter instance, removal of the hyperplastic (adenomatous) growth in the usual way will leave behind the infected remnants of the functioning prostatic glands. Persistent urinary infection fol-

lowing removal of the hyperplastic growth in the prostate may thus be explained in some instances.

When it becomes desirable to remove the infected remnants of the functioning prostatic glands, this may be adequately done by a subtotal perineal prostatectomy. These glands are so intimately connected with the prostatic capsule that removal of both together can only be accomplished by the perineal approach. The method of subtotal perineal prostatectomy by which the prostatic capsule, compressed prostatic glands, and the hyperplasia, if present, are removed together and the urethra approximated to the bladder neck is described in detail elsewhere.

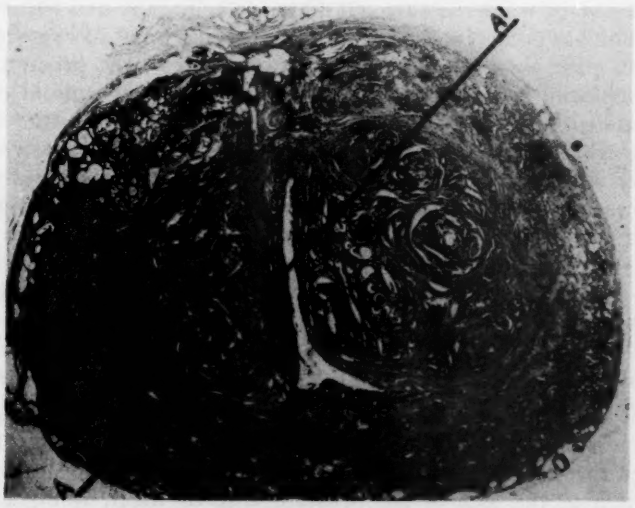

Fig. 2. Photomicrograph x 2. Cross section of prostate, showing how fibromuscular masses on each side of urethra have been entirely replaced by glandular hyperplasia. Note increased compression of true prostatic glands toward capsule compared to fig. 1; also, increased anteroposterior elongation of urethra compared to fig. 1. An urethrogram, taken in a semilateral position, denoted by line A—A', demonstrates the anteroposterior elongation of the prostatic urethra from which one may estimate the size of the hyperplastic tissue replacing the lateral prostatic lobes.

Carcinoma of Prostate

Carcinoma of the prostate has been discovered more frequently in recent years. Rich, Moore, and Deming in routine autopsies have independently found carcinoma of the prostate in 14 to 20 per cent of all male patients past 50 years of age. Young and Alcock each report an incidence of about 20 per cent of malignancies in routine prostatectomies. Many of these are not diagnosed clinically and are only found on pathologic examination.

Almost 75 per cent of prostatic malignancies found at routine autopsy were present only in the posterior lobe of the prostate. The posterior lobe of the prostate is not involved in hyperplasia (adenoma). Neither can the posterior lobe be removed by suprapubic prostatectomy or transurethral resection of the prostate. Because of its location under and distal to the verumontanum, it is compressed toward the capsule with the functioning glands of the lateral lobes during hyperplastic enlargement of the prostate.

The posterior lobe can adequately be removed by subtotal perineal prostatectomy. Removal of the prostatic capsule, to which the posterior lobe is intimately adherent, together with any enlargement of the prostate, should remove many carcinomas of the prostate at a stage when they are not diagnosed clinically and before any extension has occurred. When we consider that 1 out of every 5 to 7 patients past 50 years of age has a carcinoma of the prostate, are we not justified in removing the posterior lobe and capsule of the prostate with the obstructing portion of the gland? Any other method of treating carcinoma of the prostate is rarely curative and usually is done for palliation alone. Until the time arrives when our treatment of carcinoma of the prostate is more satisfactory in curing patients and preventing the subsequent suffering, it may not be unreasonable to consider the advisability of removing this posterior prostatic lobe and capsule when surgery must be instituted anyway for obstruction.

Determining the Nature and Extent of Prostatic Disease

It is our practice to study thoroughly the type of prostatic disease present in a specific case before deciding upon the best surgical procedure for that patient. Rectal examination is very misleading in ascertaining the size of the prostate and, of itself, is inadequate for determining the need for surgical treatment. In 50 per cent of Randall's specimens of prostatic disease, no enlargement was noted by rectal examination. It must, therefore, be assumed that many patients requiring treatment of the prostate need further study than rectal examination to determine the presence and nature of prostatic disease. It is not uncommon to have a patient who has been referred by a physician state that rectal examination has ruled out prostatic disease. Subcervical lobes, median commissural hypertrophy, and fibrous bars at the vesical outlet are among the conditions which may manifest no change in the contour or size of the prostate palpated by rectum, yet may require surgery for their relief. Malignancy of the prostate may first be suspected by a small, hard nodule noted on rectal examination.

Determination of the quantity of residual urine is always done.

One may thus estimate the extent of the obstruction and whether or not surgical intervention has become urgent. Marked urinary infection of long standing may be a factor in deciding the proper surgical procedure. Cystoscopy, when indicated, will aid the urologist in selecting the type of operation in most instances.

Considerable information is obtained from cystourethrograms taken in a semilateral position. The bladder is filled with air for the first roentgenogram, partly for a dark background contrast for the urethrogram, and partly to outline the soft tissue shadow of the prostatic intrusion into the bladder. Diverticula of the bladder may be outlined. Prostatic stones, if present, are visualized. Hippuran jelly is then injected through the urethra as another roentgenogram is taken; this outlines the length and anteroposterior spread of the prostatic urethra. One may fairly well judge the location and size of prostatic enlargements in this manner. Often one may become suspicious of prostatic malignancy from a cystourethrogram. The extent of fibrous bar contractions at the vesical outlet may be seen. The type, location, extent, and character of the obstructing enlargement are determined and the proper surgical procedure decided upon.

Selecting Type of Surgery

All of the commonly utilized types of prostatic surgery have their place, but, whenever possible, one should select the procedure which will remove the obstruction and relieve the symptoms promptly and permanently. In the past, we have treated slightly more than 60 per cent of prostatic obstructions by transurethral resection. Our most distressing complication has been persistent dysuria with infection, in some instances, lasting for several months. Urethral stricture and incontinence have occurred. Rather marked morbidity may follow transurethral resection improperly done or when portions of infected prostatic remnants are left in the prostatic urethra.

Suprapubic prostatectomy requires less skill than either transurethral resection or perineal prostatectomy. A large intravesical hyperplasia may be enucleated suprapubically with ease and, when no residual prostatic lesion is left behind, this offers relief from urinary obstruction. On the other hand when patients are not properly selected for this procedure many difficulties may be encountered. Most of us have seen surgeons struggle to remove a fibrous infected prostate suprapubically, ending up with a few bits of prostate which have been cut or pulled out because no line of cleavage existed.

Perineal prostatectomy requires skill in the approach to the prostate, as well as in its removal and the subsequent repair of the prostatic urethra. Perineal prostatectomy, improperly done, may result

in urinary incontinence or a rectourethral fistula. Training in the proper approach to the prostate, together with newer methods of repair of the prostatic urethra and bladder neck, should make these complications less frequent. Patients should be warned of the possibility of sexual impotence when subtotal perineal prostatectomy is performed.

Competence to do the surgical procedure decided upon is, of course, essential. However, selection of the proper surgical procedure in a given case is also very important, if one hopes for relief of symptoms and prolongation of life. Discrimination in selecting the procedure best suited to the individual patient should be our aim.

Subtotal Perineal Prostatectomy

New methods of perineal surgery have recently been devised. Belt's perineal approach to the prostate has been a factor in reviving interest in perineal surgery on the prostate. Gibson has advised suturing the bladder neck to control hemorrhage rather than resort to packing or one of the various types of prostatic bags.

In performing a subtotal perineal prostatectomy, the prostate may be approached by Young's or Belt's method. The prostatic urethra is opened transversely near the apex of the prostate. This incision is carried around each side, including the prostate, capsule, and urethra, severing these from the distal urethra proximal to the external vesical sphincter. The entire prostate with the prostatic urethra and capsule is removed en masse. The seminal vesicles and vasa deferentia are severed as they enter the prostate and the distal ends tied. Sutures are placed in the bladder neck to control hemorrhage. A catheter is inserted through the urethra and sutures approximate the bladder neck to the distal cut end of the urethra. Packing is rarely required. Wounds heal more promptly in most instances and patients leave the hospital earlier than is the case when hyperplastic tissue alone is removed.

By this subtotal method the obstructing tissue may be completely removed as well as all remnants of compressed prostatic tissue. No focus of infection remains in the prostatic urethra to cause persistent urinary symptoms. The posterior prostatic lobe is removed and hence a possible source of undiagnosed malignancy is eliminated. The patient should be told that temporary or even permanent sexual impotence may result from this procedure.

Conclusion

In conclusion, it seems to us logical that one must have a clear conception of the pathologic variations encountered in prostatic disease before one can decide upon the rational surgical procedure to

be employed in any given case. The point of origin and progress of the various types of prostatic disease should be constantly before us while the patient is being investigated before the surgical approach and procedure are determined. If one attempts to treat all prostatic diseases by a single surgical method, many patients will not receive the treatment best suited to them. Many patients believe that both from their own and the surgeon's viewpoints, transurethral surgery is the simplest procedure for securing relief. However, when cases for transurethral operation are improperly selected, the morbidity following this procedure may require these patients to seek further relief. In some of these patients infection of the remaining prostatic glands causes persistent dysuria, while in others an undiagnosed malignancy may remain in the compressed posterior lobe and capsule. We, as surgeons, are rightly to be considered inadequate if, through lack of preliminary investigation, study, and understanding of the pathology present, we fail to approach our problem by that method which is particularly fitted to give adequate permanent relief.

SUMMARY

1. The pathology of prostatic disease is frequently overlooked in selecting the surgical procedure for relief of symptoms.

2. Each of the three main methods of prostatic surgery has its place, and none should be used to the exclusion of the others.

3. A method of subtotal perineal prostatectomy is described.

4. Infection of the remnants of the functioning prostatic glands, prostatic calculi, and early malignancy, particularly in the posterior lobe, may be completely removed only by the perineal approach.

5. Patients should be warned of the frequent occurrence of impotence following subtotal perineal prostatectomy.

6. Recent improved surgical technic and mastery of the procedure should dispel the fear of urinary incontinence or fecal fistula.

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L. MINOR BLACKFORD, M.D.
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THE TEXAS SURGICAL SOCIETY

Important Announcement

It gives us great pleasure to announce that the Texas Surgical Society celebrated its twenty-fifth birthday on April 2, 1940, by adopting THE SOUTHERN SURGEON as its official organ. This action was not done until after the scientific sessions had been completed and so the speakers had not had an opportunity to get their talks in shape for publication. We hope to begin publishing them at an early date.

PRESENT TREND IN GRADUATE TRAINING FOR SURGERY

The C. Jeff Miller Memorial Lecture

J. M. Mason, M. D.

Birmingham

I ESTEEM it a great honor, as well as a great privilege, to have been invited to deliver the C. Jeff Miller Memorial Lecture. It is an honor to be given so prominent a place on the program of The Southeastern Surgical Congress, and a privilege to be permitted to say a few words in memory of my friend Jeff Miller, to whom I always felt very close. My acquaintance with him began when he first reached New Orleans. He was young, unknown, ambitious, and came in competition with a brilliant coterie of colleagues, some of his own age and some older, who were entrenched in directing the medical, surgical and professional affairs of the city, and had been for many years.

It is a credit to members of the Tulane faculty and to the Boards of Control of the various hospitals that they recognized that this young outsider was a man of promise, and made a place for him.

It is to his everlasting credit that he made good on every step of the way to success, and that at the time of his death he was Head of the Department of Gynecology in the School of Medicine of Tulane University of Louisiana, Senior Attending Gynecologist to the Charity Hospital and to Touro Infirmary, and was one of the outstanding civil leaders of the City of New Orleans.

During his career he was elected, successively, to the presidency of the Southern Surgical Association in 1922, of the American Gynecological Society in 1928 and of the American College of Surgeons in 1930.

From its inception he was deeply interested in The Southeastern Surgical Congress and was active in its organization in the State of Louisiana. At the 1935 meeting of the Congress he was elected president to serve for 1936, and held that office at the time of his death, March 21, 1936.

Always progressive, always forward looking, I feel that if he were with us this evening, he would be active in shaping and directing the movement about which I shall speak to you: "Present Trend in Graduate Training for Surgery."

Delivered at the Eleventh Annual Assembly of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.

Present Trend in Graduate Training for Surgery

The present demand for more extended graduate training for surgery and its related branches is the result of pressure from several directions. The underlying demand comes from the steady progress along all educational lines; the immediate impelling demands come, in the main, from the calls upon hospitals for better equipment and a higher quality of professional service, and from the multiplication of specialties.

Trend in graduate training must, therefore, be considered in connection with hospital standardization, increasing educational requirements, and the specialty boards. So exacting have these requirements become in very recent years, that many hospitals have been taken somewhat unawares, and we have been put on notice by Dr. W. C. Rappleye, President of the Association of American Medical Colleges, in his 1939 presidential address: that

> The training of a sufficient number of specialists to meet the needs of the country according to the standards of the American Boards, the Advisory Board for Medical Specialties, and the Council on Medical Education and Hospitals of the American Medical Association can be accomplished, in many instances, only by modifications in existing intern services and the development of new facilities and opportunities in the hospitals.

The progress of medical education in the United States has been steady, but sometimes slow. Starting with only four medical schools in the country at the beginning of the Nineteenth Century, we passed through the stage of apprenticeship in the study of medicine; through the period of the six weeks lecture course in each of two years; through the three year courses of five or six months each; through the American Medical Association's purge of the proprietary schools, and had settled down to the four year session with rigid entrance requirements, only to face the question of graduate training and the specialty boards.

Some years ago it had become apparent that an investigation of the hospital situation was needed, and that the practice of surgery should be placed on a higher plane than it then occupied. In 1913, the American College of Surgeons was organized with both these objectives in view.

Regarding hospitals it was accepted as axiomatic that any institution that undertook the care of the sick must be so equipped physically and so staffed professionally and ethically, as to be able to carry out its obligations to those who sought its aid. After careful investigation, a "Minimum Standard" of requirements was adopted, which had to be met before the hospital would receive the "Approval" of the College.

This resulted in immediate, far reaching and continuous improvement in hospital service.

The first inspection of hospitals of 100 beds and over was made in 1918: 692 hospitals were surveyed and 89, or 12.9 per cent, were approved. In 1938, in the same class of hospitals, 1,850 were inspected and 1,726, or 93.3 per cent were approved, either fully or provisionally.

At the time of its organization the College was the first society in America to undertake the granting of a distinguishing degree in surgery and likewise was the first to appeal directly to surgeons to elevate the standards of surgical practice.

Article 11 of the By-laws is as follows:

OBJECT—The object of the College shall be to elevate the standard of surgery, to establish a standard of competency and of character for practitioners of surgery, to provide a method of granting fellowships in the organization, and to educate the public and the profession to understand that the practice of surgery calls for special training, and that the surgeon elected to fellowship in this College has had such training and is properly qualified to practice surgery.

In time the College was subjected to the criticism that it had become too lax in applying these requirements to candidates for fellowship and for granting the title F. A. C. S.

This criticism was brought forcefully to the attention of the profession at the 1935 meeting of the American Surgical Association, by Edward Archibald, in his presidential address entitled "Higher Degrees in the Profession of Surgery." He compared the method of the American College of Surgeons for conferring the title F. A. C. S. with the methods obtaining in England, Australasia, Scotland, Canada, France and Germany for conferring higher degrees, and said:

The Board of Regents of the College, at the beginning, had to make their choice between following the example of the English College of Surgeons, with its notoriously stiff examinations, and adopting some less rigid system which would chiefly take into account a candidate's practical ability in surgery, without troubling much about theory; which, in particular, would not require of him a written or oral examination in either primary or final branches. They chose the latter plan. In view of the deplorable conditions existing in America at that time, respecting the prevalence of incompetent surgery, it seemed better to the Regents to try to elevate the standards of the general mass of surgeons than to establish by severe tests a *corps d'elite*. The majority of us will, I think, agree that at that period their decision was wise.... There was, in some degree, an emergency to be met.

His address was followed by papers on graduate and undergraduate training for surgery by Elliott Cutler (Harvard), George Heuer (Cornell) and Allan O. Whipple (Columbia); and such distinguished surgeons as Bevan, Cheever, Thomas G. Orr, Gallie,

Dean Lewis, Eugene Pool, Rodman, McClure, Hugh Cabot and Evarts Graham, took part in the discussion.

These papers and the discussions which they precipitated, proved to be the most potent influence in elevating surgical standards since the first step; which was taken by the College in 1913.

The sentiment of the meeting crystalized on two ideas: First—That laxity in scrutinizing the fitness of candidates for fellowship should be corrected by the College, and that its requirements for admission should be raised. Second—That it was advisable to organize an American Board of Surgery along the lines already approved by the American Medical Association for other specialty boards.

Accordingly, the American College of Surgeons set about to correct the conditions which had subjected it to criticism, and the American Board of Surgery came into being in 1937.

Specialty Boards

The Annual Report of the Council on Medical Education and Hospitals of the American Medical Association is found in the *Journal* of August 26, 1939. Of particular interest is that section devoted to approved examining boards on specialties.

The American Board of Surgery, organized in 1937, is composed of representatives from seven surgical associations. The component societies, with their representatives, are as follows:

	No. Members
The American College of Surgeons	3
The Surgical Section of the American Medical Association	3
The American Surgical Association	3
The Southern Surgical Association	1
The Western Surgical Association	1
The Pacific Coast Surgical Association	1
The New England Surgical Society	1
Total	13

While the examinations of this board are very rigid, and while, in addition to the internship, five years of graduate study is exacted of all applicants for certification, the range of permissible graduate study is rather elastic. The five years may be spent in one or more hospitals; or, a more varied type of instruction may be followed; a suggested schedule consisting of one year in a graduate school giving special attention to basic sciences; two years in a surgical residency, and two years assistantship to a surgeon of recognized standing. Such a combination presents many advantages to those who are unable to remain in hospital work for the full five years.

Other boards of the major surgical specialties also require extended graduate training in addition to the internship: for example—the Board for Obstetrics and Gynecology requires three years residency or service with a qualified preceptor; that for Orthopedics requires three years concentrated instruction in Orthopedics, including a residency of two years, and two years in actual practice; that for Urology requires three years study in clinics, dispensaries, hospitals, or laboratories competent to provide satisfactory training in the special field of urology.

The present graduate training requirements of the American College of Surgeons are that the applicant shall have had three years of hospital service in one or more accredited hospitals, of which two shall have been spent in training in surgery in hospitals approved by the College.

The Bulletin of the American College of Surgeons, January, 1940, contains a symposium on Graduate Training for Surgery, in which a number of outstanding teachers of surgery took part. Practically all phases of graduate instruction were discussed. No fixed plan of organization for graduate instruction has been laid down by the College but in this number of the Bulletin, and also in the April 1939 number, are given the plans of several hospitals which have received the endorsement of the College. The January Bulletin also contains an analysis of the 1939 report on "Graduate Training for Surgery," by Dr. Harold Earnheart, Assistant Director of the College. From this report we learn that 179 participating hospitals turn out annually 580 men who have completed at least two years' training in surgery; 209, or 36 per cent, have had two years training; 371, or 64 per cent, have had three or more years; and 62, or 12 per cent, have had four or more years training. He makes the comment that "The trend rapidly approaches the three year training period as essential or ideal if one is to accept the present hospital practices as the logical standard."

Concerning the bed capacity of the hospitals which have approved plans for training he says:

> Only 15 approved plans for training exist in hospitals of less than 200 beds; while there are 152 approved plans in hospitals of 200-500 beds. However, the greater burden of graduate education in surgery is carried on in hospitals of over 500 beds. Perhaps the possibility of future expansion rests principally in smaller hospitals.

He next brings up the question of basic science and says:

> We may now consider the problem of graduate education in surgery about which there is the most widely diversified opinion, namely—basic science activity. There are some who feel that this subject has been given too much prominence, that the necessity for spending any considerable time in basic science study in

graduate training for surgery is a definite reflection on a deficiency in the undergraduate system of medical education.

When the committee first began to study this subject, it seemed quite simple. However, as the work progressed, it soon became apparent that any attempt to standardize recommendations on what should constitute basic science activity in a graduate training program was impossible. It has been possible, however, to recognize three major types of basic science activity in hospitals; namely—The practical study of surgical pathology, which activity any hospital contemplating a graduate training program should be able to provide. Second—Basic Course—a more or less formal academic type of instruction such as has been developed in certain of our graduate schools of medicine. Third—Research, which activity is usually confined to highly organized hospitals which are the principal teaching units of medical schools.

No doubt there will remain much controversy over what constitutes desirable basic science activity in graduate training for general surgery and the various surgical specialties. No unreasonable or arbitrary requirements have been or will in the future be established in this graduate training program.

One can scarcely refrain from entering this controversy on the present occasion. However, I will content myself with reflecting that basic science is the foundation upon which rests the applied science of the clinic and the operating room. The student should have been well grounded in basic sciences before he left college to enter upon the clinical phase of his education. The intelligent intern or resident will keep abreast of progress in those basic sciences that directly affect his daily work by collateral reading, by discussions in ward rounds and staff conferences and with laboratory workers, and should not be subjected to further teaching of an academic type. In pathology, the situation is quite different. Nowhere in the medical curriculum can this subject be taught so comprehensively as in the hospital. Here the intern and the resident can correlate physical examination with laboratory findings; and clinical diagnoses are confirmed or disproved by study of specimens removed at operation or obtained by autopsy. As Earnheart has said, this activity any hospital contemplating a graduate training program should be able to provide.

The demands which have been made by the College, the American Medical Association and the specialty boards have added greatly to the responsibilities of the hospitals. A few years ago hospitals were besieged with applications for internships. Now there is an additional plea for residencies; and where formerly a year or two was sought, now three or more years are demanded.

Admitting the superior position now held by university hospitals and by hospitals of large bed capacity, I am by no means ready to concede that smaller hospitals cannot continue to give satisfactory

graduate training in the future, as they have in the past. Rappleye, Ochsner, and Earnheart have stated, in effect, that modifications in existing intern services and the development of new facilities and opportunities in hospitals will be necessary to meet the needs of the country according to the standards which are now being established. I have every confidence in the ability and the willingness of hospitals to make necessary modifications and to provide expanding opportunities.

My own feeling in regard to graduate training for surgery is that the intern period should be on a rotating basis, and that it should be for two years rather than for one. Many interns will not seek residencies, and the added experience gained by a two years service will vastly increase their usefulness when they enter general practice. For the intern who aspires to a residency in surgery or one of its major specialties, the two year service is equally desirable. A great part of his rotating service will be spent in some type of surgical work, and he should be given credit for this when applying to special boards for certification. Further, we should not get too far away from the old conception of the proper preparation for specialization: namely—that the specialist should not begin to limit his field until he had obtained a general view of medicine as a whole. Looking at the long period of hospital training required by the specialty boards, one asks the question, where will the facilities for these residencies be found? Earnheart tells us that now the "greater burden of graduate education in surgery is carried on in hospitals of over 500 beds," but expresses the opinion that the "possibility of future expansion rests principally in smaller hospitals."

A responsibility rests upon every hospital large enough for surgical residencies to lengthen the period of service to comply with the present demands; to the end that there will be a sufficient number of places to provide for those ambitious young men who seek them.

Since the Federal Government proposes to build a number of small hospitals throughout the country, the following quotation from the Bulletin of October, 1939, may be of interest:

> The standardization program of the American College of Surgeons is of prime importance to the small hospital which takes care of a large proportion of patients who are hospitalized. The fact that approximately 30 per cent of general hospitals of 25-50 bed capacity and 70 per cent of those from 50-100 beds in the United States and Canada have places on the approved list of the College, proves that the small hospital realizes its importance and is doing everything possible to better its condition and the service it offers. Moreover, the approval of so many of the smaller hospitals indicates their ability to meet the requirements of standardization.

Every surgeon in this audience is engaged in hospital work of some type, and to you I make this appeal:

If your hospital is small, see to it that it is on the "approved" list of the American College of Surgeons;

If large enough for internships, see that it receives the endorsement of the American Medical Association;

If still larger, see that it meets the requirements for residencies of all organizations and all specialty boards;

Above all things, see that the training you give is such that those who go out to enter practice will render service which will be a credit to your institution and to those organizations that are endeavoring to elevate surgical standards in America.

THE SAMUEL D. GROSS PRIZE

The Philadelphia Academy of Surgery for a number of years has awarded a quinquennial prize of a substantial sum of money in honor of the late Dr. Samuel D. Gross, of whom Dr. Sanders wrote so eloquently in the April number of THE SOUTHERN SURGEON, emphasizing that "he keenly recognized the need of surgeons for a thorough grounding in pathology and physiology." This prize is given to the author of the finest surgical research presented. In 1935 it went to Dr. Owen H. Wangensteen, of Minneapolis, for "The Therapeutic Problem in Bowel Obstructions."

The Gross Prize for 1940, we are delighted to note, has been awarded to Dr. Frederick Fitzherbert Boyce, of New Orleans, a Fellow of The Southeastern Surgical Congress, for his monograph "The Role of the Liver in Surgery." It gives us the greater pleasure in that some of the preliminary studies for this work were published in the SURGEON.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

DISEASES OF THE GALLBLADDER AND BILE DUCTS. By WALTMAN WALTERS, B. S., M. D., M. S. in Surgery, Sc. D., F. A. C. S., Head of Section in Division of Surgery, The Mayo Clinic; Professor of Surgery, The Mayo Foundation (University of Minnesota); and ALBERT M. SNELL, B. S., M. D., M. S. in Medicine, F. A. C. P., Head of Section in Division of Medicine, The Mayo Clinic; Professor of Medicine, The Mayo Foundation (University of Minnesota). 645 pages, with 342 illustrations on 195 figures. Price, $10. Philadelphia and London: W. B. Saunders Company, 1940.

This comprehensive up to date book by a well known gastroenterologist and beautifully adept artist surgeon is based on the extensive experience which the Mayo Clinic has had with diseases of the gallbladder and bile ducts. The opening chapter, an adequate yet pleasantly brief historical account, is followed by a review of the anatomy and physiology of the gallbladder. All forms of disease of the gallbladder and bile ducts are discussed at considerable length with some repetition which serves to emphasize the more important points. A chapter dealing with the differential diagnosis of conditions associated with jaundice is excellently presented and contains information essential to the proper management of the jaundiced patient.

Indications for operation are enumerated and the surgical procedures employed in handling diseases of the gallbladder and bile ducts are so thoroughly discussed and clearly illustrated that they serve as an excellent guide to the surgeon who wishes to duplicate them. Preoperative and postoperative management is considered in minute detail and special attention is given to the jaundiced patient and the use of vitamin K in controlling the hemorrhagic diathesis. There are also helpful chapters on cholecystography, cholangeography, dietetics, methods of determining the prothrombin time, and symptoms which may follow cholecystectomy or other operations on the gallbladder.

This book is too advanced for the average medical student but a knowledge of its contents is essential for the internist or surgeon who treats diseases of the gallbladder and bile ducts. It represents a noteworthy attempt to advance our knowledge of gallbladder diseases and should be a very popular book.

GEORGE F. ARCHER, M. D.

FRACTURES, DISLOCATIONS AND EPIPHYSEAL SEPARATIONS. By HARRY C. W. S. DE BRUN, M. D., F. A. C. S., Adjunct Professor of Surgery, New York Polyclinic Medical School and Hospital; Associate Visiting Surgeon, Swedish Hospital, Brooklyn; Consulting Skeletal Surgeon, New York Police Department; Member Association Francaise de Chirurgie. 468 pages, with 150 illustrations. Price, $5. Chicago: The Yearbook Publishing Company, 1940.

A concise, authoritative handbook such as this should be very valuable to the general practitioner or general surgeon in the intelligent management of fractures and dislocations. G. F. A.

HANDBOOK OF ORTHOPAEDIC SURGERY. By ALFRED RIVES SHANDS, JR., B. A., M. D., Medical Director of the Nemours Foundation, Wilmington, Delaware; Associate Professor of Surgery in Charge of Orthopaedic Surgery, Duke University School of Medicine, Durham, North Carolina (On Leave of Absence). In Collaboration with RICHARD BEVERLY RANEY, B. A., M. D., Associate in Orthopaedic Surgery, Duke University School of Medicine. Illustrated by JACK BONACKER WILSON. 567 pages, with 154 illustrations. Price, $4.25. St. Louis: The C. V. Mosby Company, 1940.

The first thing that strikes one on taking up this book is that a second edition was necessary in less than three years. This is rather unusual for a first book by a young man,—and Dr. Shands is still two years younger than we are. This speaks well for the excellence of the original plan which was to present an outline of orthopedics with the most approved methods of treating the disorders in this field. The second edition has been extensively revised, and includes a bibliography of important work published in English up to July 1, 1939.

Shands and Raney consider every condition (except recent fractures) that can be listed under the head of orthopedics. They discuss hemophilia and torticollis, various diseases of the nervous system, including the early stages of poliomyelitis, the various types of arthritis and congenital deformities, endocrine dyscrasias and march foot, and many things that will prove unfamiliar to most of us. In covering so much ground, however, they waste no words and yet they write in a style that is clear and simple.

The book does not attempt to provide sufficient detail to enable the general surgeon to carry out expertly certain orthopedic procedures, but it does give him the principle involved. In other words, it is designed primarily for the undergraduate student and, in our opinion, it is an ideal textbook.

One cannot leave this handbook without commenting on its neat general appearance. Most of the illustrations are line drawings by a clever artist: those that were borrowed from other authors have been redrawn. Even x-rays have been reproduced as drawings. The avoidance of photographic illustrations makes possible the use of a dull white paper which sets off the beautiful work of Mr. Wilson.

SHOCK. BLOOD STUDIES AS A GUIDE TO THERAPY. By JOHN SCUDDER, M. D., Med. Sc.D., F. A. C. S. From the Surgical Pathology Laboratory of the College of Physicians and Surgeons, Columbia University, and the Department of Surgery, the Presbyterian Hospital, New York City. 323 Pages. 55 Illustrations. Five plates (three of which are in color). Price, $5.50. Philadelphia: J. B. Lippincott Company, 1940.

This book represents a praiseworthy attempt to increase our knowledge of the blood changes associated with shock. The treatment of shock by heat and the intravenous administration of blood and saline is well recognized, but there remains in the minds of many only a vague concept of the underlying blood changes associated with the phenomenon of shock.

Dr. Scudder opens his book with a discussion of the various theories of shock, the vasoconstriction and peripheral hemoconcentration often seen in shock, and a thorough discussion of potassium in reference to its distribution, assimilation, action on the neuromuscular and blood vascular systems, and the changes in its concentration in whole blood and plasma during shock.

Twenty-eight cases of shock are tabulated, giving the clinical course, treatment, and results of the following blood studies: capillary blood specific gravity, hematocrit determination of venous blood, plasma specific gravity, plasma proteins, whole blood potassium, calculated cell potassium, and plasma potassium. It is interesting to note that a preliminary increase in the specific gravity of the peripheral blood may herald by several hours the falling blood pressure of shock.

Twenty-seven cases in which adrenal cortical extract (eschatin) was used in conjunction with 5 per cent saline in the treatment of shock, hemorrhage, burns, intestinal obstruction, and acute pancreatitis are tabulated. The idea is expressed that cortical extract and saline are definitely beneficial in such cases. Of special interest are cases of burns from the Hindenburg disaster.

The book closes with a listing in chronologic order of historical developments in the conception and treatment of shock, in the physiologic and toxicologic effects of potassium, and in the functions of the adrenal glands.

The bibliography is voluminous. A brief laboratory manual will be of particular help to those wishing to duplicate the author's tests for specific gravity of blood and plasma.

The material for this book is admirably selected and prepared. It will be interesting to those practicing surgery in close association with a medical center. However, while this reviewer feels that a thorough understanding of blood changes in shock is essential, he suspects that financial and technical reasons will prevent, at least for the present, the widespread use of many of the tests herein advocated by the author.

GEORGE F. ARCHER, M. D.

TEN YEARS IN THE CONGO. By W. E. DAVIS. 301 pages. Price, $2.50. New York: Reynal & Hitchcock, 1940.

Many doctors seek relaxation in murder mysteries, but they can find an even greater change from their everyday life in this unadorned but fascinating account of the Congo. In passing it is a relief to know that the atrocities reported thirty years ago in the muckraking magazines are no longer being perpetrated there.

After serving with the Marines in the World War, Bill Davis studied medicine and spent ten years as a missionary in Africa. He is now enjoying a large general practice in Kentucky. Only a real doctor with a sense of humor could have penned this book. He is the kind of fellow the reviewer would like to know. However (with apologies to Mr. Herford),

I've never been to Congoland
I hope I'll never see there.
I'd rather read of Afric strand
A million times than be there.